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A CONTRIBUTION TO THE KNOWLEDGE OF PRIMARY DUODENAL CANCER[1]

by

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Since HAMBURGER described the first case of primary duodenal cancer in 1746 an extensive literature has gradually appeared on this disease. The roentgenological picture of duodenal cancer has however only been sparely published, and in only a very small number of cases was it possible to make an exact roentgenological diagnosis which was later verified at operation or autopsy and by microscopical examination.

It may therefore be of interest to publish a record of another two cases of duodenal cancer, in which the radiographical examination led to the diagnosing of the disease, and where the roentgenological picture was found to coincide with the patho-anatomical findings at the operation in one case, and at autopsy in the other case.

That primary duodenal carcinoma is a very rare disease is evident from a number of different statistics published. In an autopsy material comprising 350,286 cases EGER gives the figure of 0.033 per cent. And BRILL estimates the frequency of duodenal cancer at 0.3 per cent, of all intestinal carcinomas.

The essential condition for performing the roentgenological examination is that the radiologist is fully acquainted with the various forms in which duodenal cancer can appear. (Without this knowledge it will not be possible for him to judge the roentgenological picture correctly.)

In regard to the different parts of the duodenum from which the duodenal carcinoma may originate, MATEER and HARTMAN suggested the following classification, which has since been universally adopted, 1. Supra-ampullar carcinoma, arising from the mucosa between the pylorus and the papilla of Vater, 2. Peri-ampullar carcinoma which arises from

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the papilla of Vater or the ampulla of Vater, and 3. Infra-ampullar carcinoma which has its origin in the mucosa between the ampulla of Vater and the duodenojejunal flexure. — According to these authors, the peri-ampullar localisation is the most common one, estimated by them at 6 per cent. of all duodenal carcinamos. The incidence of infra-ampullar carcinomas seems to be about half that of the supra-ampullar. All kinds of transitional forms occur, but the classification is of practical, clinical importance.

In the same manner as carcinomas in other areas of the intestinal canal, duodenal cancer also appears principally in two different forms. The most common is the soft, cauliflower-like, polypous form penetrating into the lumen of the intestine, which readily ulcerates and breaks up. A less common form is the scirrhous tumour which grows circularly in the intestinal wall, more or less severely constricting the lumen of the intestine.

In some cases the tumour grows rather superficially in the wall of the intestine and has its greatest expansion in towards the lumen. In other cases it infiltrates all the layers of the intestinal wall and occasionally metastasises to adjacent organs, most frequently to the pancreas. In the latter case the starting point of the tumour can often be ascertained only by a careful histological examination.

The common feature in all the different forms of duodenal carcinomas is their tendency to constrict the lumen of the duodenum. As the constriction increases compensatory hypertrophy occurs in the walls of that part of the duodenum situated above the obstruction. This hypertrophy is secondarily attended by a dilatation of the duodenum, the pylorus and possibly also of the stomach. If the tumour originates in the papilla of Vater or its vicinity, however, it causes in the first place, already at an early stage, a pressure on the choledochus and pancreatic ducts, attended by symptoms of stasis in the bile ducts and the pancreas. It is not until later that the tumour, if allowed to develop, brings about a constriction of the lumen of the intestine.

The histological picture of duodenal cancer is the same as that of other carcinomas of the intestinal canal. Thus the commonest type is an adenocarcinoma arising from the duodenal mucosa. The tendency to metastatic growth does not seem to be very strong, in a great many cases probably because of the rapid course of the disease, during which metastases have not time to develop.

The *symptoms* in primary duodenal cancer depend to a great extent on the localisation of the tumour in the duodenum. In the peri-ampullar form the dominating symptom is the biliary stasis, clinical symptoms of intestinal obstruction do not appear until later. In the other tumour forms the symptoms depend on the degree of constriction of the lumen of the intestine.

These symptoms most frequently begin as gastric symptoms, the patients thus complain of loss of appetite, eructations and nausea. As the tumour grows these symptoms are accentuated, vomitings occur, accompanied by wasting and obstipation. Occasionally the vomitings are copious and now and then resembling »coffee grounds», less frequently really haemorrhagic. Pain is a variable symptom. As a rule the pain is localised to the epigastrium or the right hypochondrium but occasionally to the whole of the upper part of the abdomen.

The *diagnosis* of primary duodenal cancer offers difficulties. On the one hand characteristic symptoms are absent, most of the symptoms present are common to several diseases in the upper part of the abdomen. On the other hand, owing to the great rarity of the disease, as compared with the relatively high frequency of malignant affections in adjacent organs, it is very easy to overlook the possibility of a duodenal cancer. Consequently most cases have been discovered at operation or autopsy.

The attempts made to diagnose duodenal cancer roentgenologically have also been very unsuccessful so far, which is evident from a few classifications in the literature. HOFFMAN an PACK made a clinical and roentgenological study of 5 cases of their own together with 15 cases from other hospitals. In 15 cases radiological changes were found in the duodenum, which had been recorded in 14 cases. In only 1 case was the diagnosis of primary duodenal cancer suggested, a diagnosis which was verified later. In the other cases the affection had been diagnosed as pyloric cancer, duodenal ulcer, pancreatic cancer, etc.

STEWART and LIEBER published 6 cases of *supra-papillary* cancer of their own. They also collected a further 104 cases from the literature, in only 35 of which, however, were there any satisfactory clinical data and microscopical verification. Of these 41 cases radiological examinations had been made in only 18. In 11 cases roentgenological changes were found in the duodenum, but in only 2 of these cases had primary duodenal cancer been diagnosed pre-operatively.

LIEBER, STEWART and LUND have also classified 2 cases of their own together with 28 cases of duodenal cancer with *infra-papillary* localisation, published in the literature. Altogether 62 cases were published but only the 28 cases included in their classification had been satisfactorily elucidated. Roentgenological examinations had been made in 18 of these 30 cases, but in 3 cases the examinations were unsatisfactory. No changes were recorded radiographically in 4 cases, while in 5 cases the changes observed did not coincide with the anatomical findings. In the remaining 6 cases there was an agreement between the radiological picture and the patho-anatomical changes.

The difficulty of roentgenologically recording and correctly diagnosing primary duodenal cancer is mainly due to the position of the duo-

denum with its close proximity to the stomach, the bile ducts and the pancreas. Quite a number of different affections in these organs may on the radiogram convey the impression of a primary duodenal affection.

Thus if the duodenal tumour is situated *supra-ampullarly* it may, on account of its position, cause a deformity of the duodenal cap or of the prepyloric part of the intestinal canal. This deformity may then be interpreted as being due to an ulcer in the duodenum or the pylorus or as a cancer situated prepylorically (EGER). The problem in that case will be to ascertain by means of a further control examination whether the stomach can be absolved and with the aid of the history of the case to exclude the possibilitity of a duodenal ulcer.

In a *peri-ampullar* localisation the differential diagnosis will rest between cancer in the bile ducts, cancer in the head of the pancreas and pancreatitis. In the early stages there is very little prospect of distinguishing a cancer of the bile ducts from a peri-ampullar duodenal cancer. It is not until a later stage, when the duodenal tumour has commenced to constrict the lumen, that it is possible to show roentgenologically the presence of a stenotic peristalsis, and a stasis with a dilatation of the bulb and the pylorus. These symptoms point to a primary duodenal affection, since as a rule they have no time to develop in cancer of the bile ducts.

The clinical symptoms in a cancer of the head of the pancreas may present a close resemblance to the radiological picture in both peri-ampullar and supra-ampullar duodenal cancer. This is all the more true since duodenal carcinoma in a great many cases extends secondarily to the head of the pancreas. Even at autopsy the primary source of the tumour may be difficult to establish.

It is not always that the roentgenological pictures are so clear and so easy to interpret as when the pancreatic tumour has only caused a dilatation of the curvature of the duodenum without effecting the lumen of the duodenum. More commonly the tumour produces an impression on the duodenum or, when it manifests a tendency to infiltrate, an irregularity in the setting of the mucous membrane with or without stenosis of the lumen of the intestine. In the latter case, when an infiltration of the intestinal wall is present, the primary site of the tumour cannot be established roentgenologically, unless a dilatation of the duodenal curvature can be shown at the same time, a feature that indicates an expansive process in the head of the pancreas.

Like carcinoma of the pancreas, both acute and chronic pancreatitis may bring about a deformation of the duodenal curvature as well as changes in the lumen by impression, although the changes are seldom very pronounced. Otherwise acute pancreatitis is as a rule easy to differentiate owing to its clinical symptomatology. — FROSTBERG reported a case of

pancreatitis with a deformity of the concave surface of the duoedenum, in which the differential diagnosis rested between pancreatitis and a neoplasm in the pancreas (Fig. 1). Post-mortem examination showed the presence of a pancreatitis.

Fig. 1.

Finally, as to the *infra-ampullar* localisation of the tumour, a cancer situated in the distal part of the duodenum associated with stenosis and a secondary dilatation of the curvature may, as illustrated by Case 2 below, present the same roentgen picture as in so-called arterio-mesenteric obstruction with a compression on the mesenteric root. A similar deformity of the duodenum is also produced by external pressure on the duodenal curvature due to periduodenal affections, such as enlarged retroperitoneal glands or a tumour in the adjacent organs (EGER).

Case 1. Female, aged 60. Had previously been in good health. In May, 1932, the patient had had attacks of pain in the epigastrium without any relation to meals and lasting for varying periods. Sometimes also night pains. Since September, 1932, gradually increasing attacks of nausea and vomitings. At the same time anorexia and great loss of flesh — she lost 8 kilos in 1 year. No fever.

Fig. 2.

Fig. 3.

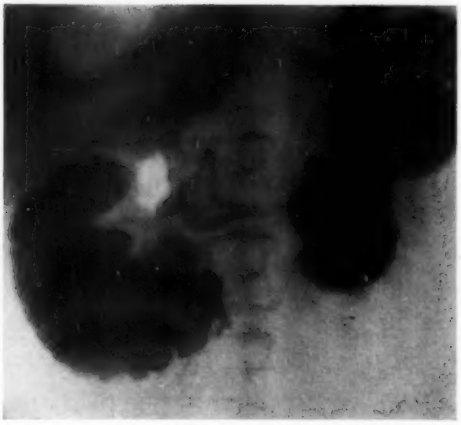

Fig. 4.

A roentgen examination of the gall-bladder, stomach, small intestine and colon made at another hospital was negative (Fig. 2). The patient was re-examined 3 months later at another hospital. The patient had for some time past had repeated copious vomitings. This examination revealed »a filled duodenum, especially the pars descendens which may indicate a duodenal stenosis, which in that case may be due to a strangulation of the mesenteric vessels». (Fig. 3.)

On February 28, 1933, the patient was again examined roentgenologically, this time at Sophiahemmet, Stockholm, where she had been admitted. The result of this examination was: »The general picture of the abdomen shows that the duodenum is distended and filled with gas. No pathological gas-filling of the intestines otherwise. Examination with contrast medium: The stomach is somewhat dilated. The bulbus duodeni, the duodenum desc. and the greater part of the duodenum inf. are greatly dilated (maximum width 7 cm.). Close to the duodenojejunal flexure the lumen narrows abruptly, becoming only about the width of a quill. The stricture is markedly concentric. — A few small drops of the contrast medium can be pressed into the stricture. The fluid does not pass the stricture in spite of the fact that the examination lasted for $1^1/_2$ hours. The stricture has an oval, ridged margin».

Fig. 5.

Summary: The roentgenological examination revealed »a greatly constricting infiltration in the duodenum close to the jejunal flexure — probably cancer» (HELLMER). (Figs. 4 and 5.)

Operation, March 1, 1933 (HYBINETTE) verified the diagnosis of a stenosing cancer in the lower part of

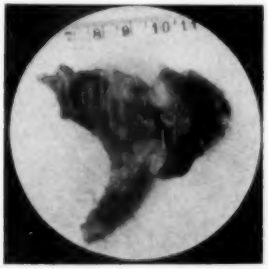

Fig. 6.

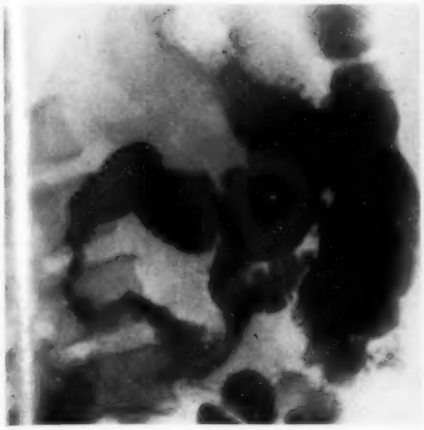

Fig. 7.

Fig. 8.

the duodenum, and a resection of the tumour was performed, after which the duodenum was sutured end to end.

Patho-anatomical diagnosis (REUTERWALL): The microscopical examination of the excised specimen showed: »A carcinoma extending through the musculature down into the surrounding tissue. In the section the carcinoma breaks off fully 1 cm. from one margin of the resection but extends almost right up to the other margin» (Fig. 6).

The patient made a good recovery after the operation and a control roentgenexamination in November, 1934, showed normal conditions in the duodenum, not even a trace of the site of resection could be seen.

Case 2. Male, aged 64. For the past 5 years the patient has had periods of flatulence and fermentations in the stomach and constipation. — Two months before admission to Lund Hospital on September 16, 1938, the disturbances changed character. The patient now had a dull aching in the epigastrium, coming on $^1/_2$ or $^3/_4$ hour after meals, irrespective of the nature of the food. This aching lasted for about 1—2 hours, during which the patient felt his stomach to be distended. Eructations and nausea also occurred, but no vomitings. The patient felt weak and tired and had no appetite. Lost 5—6 kilos in 2 months. — During the patient's stay at the hospital he was found to be affected with achylia, Weber's test being positive in all tests made. The temperature was afebrile. On October 11 the patient began to show signs of icterus, and on October 11 the bilirubin in the blood was 10/200,000.

Roentgenological examination: Cholecystography made on September 23 showed not quite an optimal contrast filling of a highly situated gall-bladder. Slow rate of emptying after a fat meal. — Cholecystography was again made on October 1: »still a very poorly contrast-filled gallbladder».

On October 7 the radiographical examination of the *stomach* and the *duodenum* revealed: »An infiltration about 5 cm. in length in the genu inf. duodeni. The infiltration protrudes into the lumen and constricts it. The right stretch of the duodenum asc. is somewhat impressed. Marked stasis in the duodenum descendens. — Thus there is evidently a neoplasm present, which involves the duodenum. It cannot be determined for certain whether this neoplasm arises from the duodenum or whether it has metastasised to the duodenum from, for instance, the pancreas» (HELLMER). (Figs. 7 and 8.)

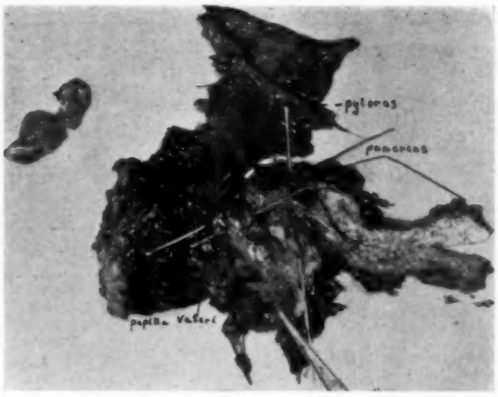

Fig. 9.

The patient was recommended to undergo an operation but he refused. He died on November 10, 1938. The last weeks before his death the patient had very copious vomitings, was greatly emaciated and had marked icterus.

Patho-anatomical diagnosis (Prof. SJÖVALL): At the juncture between the descending and terminal parts of the duodenum there is a large, deeply ulcerated tumour with medullary margins penetrating deeply into the lumen. The ductus choledochus and the ductus pancreaticus open separately into the medullary, proximal margin, both ducts are easily soundable but beyond the orifice they are both greatly dilated, the ductus pancreaticus being markedly ranular in character. The caudate part and body show on the surface and on the cut surface an entirely unchanged parenchyma. The surroundings of the head are not infiltrated by the tumour anywhere except in the direction of the duodenum. In the section the parenchyma of the pancreas also appears to be normal, and the border along the duodenal tumour, which penetrates all the layers of the duodenal wall, and still more along the lymph glands close to the head infiltrated by the tumour, also appear to be sharply defined. (Fig. 9.)

Microscopical examination: »The tumour infiltrates only that part of the head of the pancreas situated nearest the intestinal wall, and from this we get the impression that the tumour originates from the intestinal wall, grows concentrically from the site of origin, and with the tendency of a medullary cancer has greatly decomposed in the central area of its distribution».

Thus the two cases reported here were cases of primary duodenal carcinoma with infra-ampullar localisation, which had been roentgenologically described and diagnosed and microscopically verified.

In Case 1 the tumour was a scirrhus, which caused a marked stenosis of the lumen of the duodenum with signs of stasis above the stricture. The difficulty of radiologically diagnosing is evident from the history of the cases. In the first roentgenological examination the duodenal curvature was not subjected to a special investigation. But in reexamining the radiograms one finds a suspicion of a stasis in the proximal parts of the

duodenum on one of them. In the second roentgenological examination a suspected duodenal stenosis had been recorded, which was thought to be due to a strangulation of the mesenteric vessels. It was not until the third examination, when the patient was examined in different positions, especially lying on his left side, that a small quantity of the contrast medium could be pressed into the stricture, and by that means it was possible to ascertain the nature of the stricture. The subsequent operation confirmed the roentgenological picture in all respects.

It is noteworthy that although symptoms of the tumour had been manifested for nearly a year, it could be radically removed in the operation, and that no metastases could be observed. We may therefore venture to hope that there are prospects of performing a radical intervention, provided the scirrhous form is discovered early enough.

In Case 2 the radiological examination had revealed the presence of an infiltration in the duodenum inf., penetrating into the lumen and with a stasis in the duodenum descendens. The patient had been examined in different positions and with small quantities of a thick contrast medium, the delineations of the mucosa appearing very beautifully, against which the infiltration was outlined. — However, one did not venture to exclude for certain a secondary involvement from, for instance, a tumour of the pancreas, although the diagnosis of primary duodenal cancer was considered to be most probable. — At autopsy a large ulcerated tumour with elevated ridges that penetrated into the lumen was found at the spot in the duodenum indicated, and this tumour had spread to the adjacent part of the head of the pancreas. The diagnosis was not settled until the result of the microscopical examination was known.

Thus although exceedingly great difficulties are encountered in roentgenologically diagnosing a primary duodenal cancer, much better results can certainly be obtained than has been the case hitherto. In every investigation of the stomach and the intestinal canal particular attention should be devoted to the duodenum, especially its distal portion. This part of the duodenum is as a rule passed very rapidly by the contrast medium in an upright position, the medium also frequently passing this area in small portions. On that account a very slight change in the intestinal wall may very easily escape the attention of even a trained examiner.

In order to obtain a good and continuous filling of the entire duodenum the patient should be examined in different positions, especially lying on the left side, and small quantities of some kind of thick mucosal contrast medium should be used. In a simultaneous manual compression and palpation it will then be possible to obtain a very good idea of the condition of the duodenum.

By means of a careful roentgenological examination of the duodenum on these lines one may hope to contribute still further in diagnosing duodenal carcinoma.

SUMMARY

The author reports 2 cases of roentgenologically diagnosed primary cancer of the duodenum. In the first case a resection of the tumour was performed with satisfactory results, the patient was found to be symptom-free at a control examination made $1^1/_2$ years after the operation. In the other case the post-mortem examination verified the roentgenological diagnosis.

The technique of the radiological examination is discussed.

ZUSAMMENFASSUNG

Verf. teilt 2 Fälle von röntgenologisch diagnostiziertem primärem Duodenalcarcinom mit. Bei dem ersten Fall wurde der Tumour mit gutem Erfolg reseziert, bei der Nachuntersuchung nach $1^1/_2$ Jahren war der Patient beschwerdenfrei. Bei dem zweiten Fall bestätigte der Sektionsbefund die röntgenologische Diagnose.

Besprechung der röntgenologischen Untersuchungstechnik.

RÉSUMÉ

L'auteur présente 2 cas de cancer primaire du duodénum diagnostiqués par la radiographie. Dans le premier cas une résection de la tumeur fut pratiquée. Elle donna un résultat satisfaisant, un an et demi après l'opération le malade était exempt de symptomes. Dans l'autre cas l'autopsie vérifia le diagnostic radiologique.

La technique employée dans l'examen radiologique est discutée.

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US DEM KRANKENHAUS DES FINNISCHEN ROTEN KREUZES, VORSTAND PROF DR. MED. S. BROFELDT. RÖNTGENABTEILUNG, VORSTAND DR. MED. S. MUSTAKALLIO.

ÜBER DIE INSERTIONSSCHMERZEN, IHRE RÖNTGENDIAGNOSTIK UND -BEHANDLUNG[1]

von

S. Mustakallio und *H. Laitinen*

Nachdem die grosse soziale Bedeutung der rheumatischen Krankheiten in letzter Zeit erkannt und allgemein anerkannt worden ist, hat sich die Aufmerksamkeit immer mehr den verschiedenen Lokalisationen der rheumatischen Infektion zugewandt. Früher herrschte die Ansicht, dass die Intima des Herzens und die serösen Häute der Gelenke die hauptsächlichen Lokalisationsstellen dieser Infektion seien. Aber die pathologisch-anatomischen Untersuchungen letzter Zeit, von denen in erster Linie die von KLINGE erwähnt seien, haben gezeigt, dass die für die rheumatische Infektion kennzeichnenden Gewebsveränderungen in jedem beliebigen mesenchymalen Gewebe anzutreffen sind. Gewöhnlich wird die rheumatische Infektion denn auch heute als eine Allgemeininfektion des mesenchymalen Gewebes aufgefasst. Manche Krankheit, deren Ursache früher unklar war, ist auf diese pathologisch-anatomisch schon ziemlich gut definierte Infektion zurückgeführt worden. Auch die Insertionsschmerzen sind im Begriff, zur Gruppe der rheumatischen Krankheiten überzugehen.

Die Insertionsschmerzen treten im medizinischen Schrifttum erstmals eigentlich im Jahre 1909 auf. Damals beschreibt der Franzose VULLIET das Krankheitsbild der Epikondylitis klar und eingehend. Er selbst war daran bei Fechtübungen erkrankt, und als ihre Ursache betrachtet er Überanstrengung sowie auch ein direktes Trauma. Doch genügen seiner Ansicht nach Trauma und Überanstrengung allein nicht dazu, die Ursache dieser Krankheit aufzuklären, sondern es muss noch irgendeinen anderen ätiologischen Faktor geben. In der Tat sagt VULLIET, dass bei dieser Krankheit auch andere Lokalisationen vorhanden seien als der Epicondylus humeri. Nach seiner Darstellung ist nicht viel Neues zu dem Krankheitsbild hinzugefügt worden, aber die Ätiologie und das

[1] Bei der Red. am 6. VI. 1939 eingegangen.

Wesen der Krankheit sind lebhaft diskutiert worden. Meistens betrachtet man jedoch als ihre Ursache ein unmittelbar oder mittelbar auf die Insertionsstelle gerichtetes Trauma und eine bestimmte individuelle Anlage, also dieselben Faktoren wie überhaupt bei örtlichen rheumatischen Krankheiten. Ihrem Wesen nach ist die Krankheit nach den meisten Forschern ein entzündlicher Prozess an der Insertionsstelle, zunächst im Periost und in der Sehne der Insertion.

Auf der Röntgenabteilung des Finnischen Roten Kreuzes sind in den Jahren 1933—1938 47 Fälle von Insertionsschmerzen behandelt worden. Ein Fall (Abb. 1) ist uns ausserdem zuvorkommenderweise von Dr. med. A. A. SEPPÄLÄ zur Verfügung gestellt worden. Zu derselben Zeit wurden auf der genannten Abteilung über 200 Fälle von Periarthritis humeroscapularis behandelt, von denen ein grosser Teil augenscheinlich zur Gruppe der Insertionsschmerzen gehört. Diese Fälle hat der eine von uns früher in einer Untersuchung bearbeitet (S. MUSTAKALLIO 1939). In diesen Fällen besteht oft eine ganz lokale Empfindlichkeit in der Spitze des Tuberculum majus humeri, in der Humerusinsertion des M. deltoideus und in der Spitze des Processus coracoideus. Meist ist die Empfindlichkeit jedoch über ein grösseres Gebiet ausgebreitet, und die Insertionsschmerzen sind dann nur Teilsymptome der Periarthritis.

Die Fälle unseres Materials verteilen sich folgendermassen auf die verschiedenen Lokalisationen:

Epicondylus humeri	21
Calcaneus	21
Crista ilei	2
Tuberositas deltoidea humeri	1
Processus spinosus vertebrae	1
Processus styloideus radii	1

Zunächst geben wir zusammenfassend eine Tabelle, aus der die wichtigsten allgemeinen Verhältnisse unserer Fälle ersichtlich werden.

Lokalisationen	Zal der Fälle	Männer	Frauen	Durchschnittl. Alter	Geistig Arbeitende	Körperlich Arbeitende	Erkrankte Seite: Rechts	Erkrankte Seite: Links	Erkrankte Seite: Beiderseitig	Vom Pat. angeg. Ursache: Trauma	Vom Pat. angeg. Ursache: Überanstrengung	Vom Pat. angeg. Ursache: Ursache unbekannt
Epicondylus humeri	21	14	7	44.8	14	7	17	4	0	5	9	7
Calcaneus	21	16	5	32.4	8	13	7	5	9	3	4	14
Sonstige	5	0	5	43.6	3	2	1	2	2	0	0	5

Wie aus der Tabelle hervorgeht, erkranken die Männer häufiger als die Frauen an Epicondylus- und Calcaneusschmerzen. Das dürfte daran

Abb. 1. Im oberen Teil des Epicondylus lateralis eine Knochenzacke und daran anschliessend nach unten drei schwache kleine Kalkschatten. Konturen des Epicondylus im oberen Teil etwas uneben.

beruhen, dass die Männer wegen ihrer Berufe und ihrer Sportinteressen in höherem Grade Traumen und Überanstrengungen ausgesetzt sind, welche offenbar eine Ursache der Insertionsschmerzen darstellen. Bemerkenswert ist das durchschnittliche Alter der Erkrankten. Es zeigt, dass die Insertionsschmerzen in derselben Altersperiode auftreten wie die rheumatischen Krankheiten überhaupt. Die rechte Seite ist wesentlich häufiger erkrankt als die linke, was sich wahrscheinlich daraus erklärt, dass die rechte Seite öfter überanstrengt wird.

Überanstrengung und Trauma sind am häufigsten als Ursache der Krankheit angegeben. Das Trauma war in der Regel recht unbedeutend. Aus diesem Grunde besteht auch ein Missverhältnis zwischen Ursache und Wirkung, denn es ist schwer zu verstehen, wie eine Überanstrengung der Muskeln und ein kleines Trauma eine so langdauernde Krankheit hervorrufen können. So ist in einem grossen Teil der Fälle keine derartige äussere Ätiologie festzustellen. Darum und weil die Krankheit recht typisch ist, muss man annehmen, dass es eine gemeinsame eigentliche Ursache gibt und dass das oft in der Anamnese vorkommende Trauma nur

Abb. 2. Auf der Unterfläche des Tuber calcanei kleine atrophische Flecken und an seiner Vorderfläche ein kleiner atrophischer Sporn. Im unteren Bild wird die Spitze des Spornes von einem getrennten Kalkschatten gebildet.

als ein auslösender Faktor zu Ausbruch der Krankheit zu b trachten ist. Das Krankheit bild erinnert in hohem Grad sowohl in bezug auf seine En stehung als seine Symptome a örtliche rheumatische Krank heiten, wie an die Periarthrit den. Offenbar gehören auc die Insertionsschmerzen zu de rheumatischen Affektione Dafür spricht auch die Tat sache, dass in 8 Fällen unsere Materials auch andere Symptome einer allgemeinen rheumatischen Infektion vorkommen, z. B. Gelenkentzündungen. In Fällen, die grössere Knochenveränderungen im Röntgenbild gezeigt haben, sind WaR und Kahn negativ gewesen.

Die in die Epikondylen verlegten Insertionsschmerzen werden gewöhnlich als *Epikondylitis* bezeichnet. Meistens ist die Krankheit in den lateralen, nur selten in den medialen Epicondylus lokalisiert. In unserem Material sind nur zwei Fälle vom medialer Epikondylitis vertreten. Die Epikondylitis entsteht meist bei der Ausübung solcher Sportarten, bei denen die Extensoren der Hand und der Finger angestrengt werden, wie beim Tennis- und Golfspiel sowie beim Fechten. Die Extensoren inserieren mittels einer gemeinsamen Sehne an dem lateralen Epicondylus des Humerus. An dieser Ansatzstelle entsteht ein periostaler Reizzustand, der sich in Schmerz, Druckempfindlichkeit, oft in örtlicher Anschwellung und bei Bewegung auftretenden Schmerzen äussert. Die Schmerzen sind vielfach so heftig, dass sie die Nachtruhe des Patienten stören, und sie machen ihn zusammen mit der Empfindlichkeit arbeitsunfähig oder er ist wenigstens gezwungen, seinen Lieblingssport für lange Zeit einzustellen. In unseren Fällen war die durchschnittliche Dauer der Krankheit vor der Röntgenbehandlung etwa

Abb. 3. Auf der Unterfläche des Tuber calcanei kleine atrophische Flecken und ein paar Zackenbildungen.

Abb. 4. Auf der Unterfläche beider Calcanei eine schmale atrophische Zone mit kleinen Zacken und Unebenheiten. Im oberen Bild setzen sich die Veränderungen in das Gebiet der Hinterfläche fort.

Monate. Eine so lange, wenn auch nur partielle Arbeitsunfähigkeit verdient sozial Beachtung.

An *Fersenschmerzen* leiden meist Plattfüssige. In der Regel sind die Schmerzen bei dem Tuber calcanei lokalisiert, an dem die kurzen Flexoren

Abb. 5. An der Ansatzstelle der Achillessehne nach oben gerichteter Knochensporn mit scharfen und glatten Grenzen.

der Fusszehen, das Ligamentum plantare longum und die Plantaraponeurose inserieren. Manchmal ist die Schmerzregion jedoch viel ausgedehnter, oft sind auch die Hinter- und Seitenflächen der Ferse empfindlich. An dem Tuber calcanei findet man im Zusammenhang mit den Schmerzen auch oft eine Sporenbildung. Seltener hat die Krankheit ihren Sitz an der Insertionsstelle der Achillessehne. Als Symptome erscheinen Druckempfindlichkeit und Fussschmerzen beim Stehen und Gehen. Die Krankheitsdauer ist auch hier sehr lang. In unseren Fällen war sie durchschnittlich ca. 7 Monate. Ja, einige Patienten sind mehrere Monate völlig arbeitsunfähig gewesen.

An anderen Lokalisationen finden sich in unserem Material 5. Die Schmerzen bestanden in 2 Fällen in den Insertionen der langen Rückenmuskeln an der Crista ilei nahe bei den Artic. sacroiliacae. Die Symptome waren auch da Druckempfindlichkeit und Schmerzen beim Bücken und

Abb. 6. Links auf der Oberfläche der Crista ilei, an das Sakroiliakalgelenk grenzend ein Knochenvorsprung. Rechts an der entsprechenden Stelle ebenfalls ein kleiner Knochenvorsprung.

beim Strecken des Rückens. Andere Lokalisationen lagen in vereinzelten Fällen vor, mit dem Sitz im Processus styloideus radii, in der Humerusinsertion des M. deltoideus und im Processus spinosus des vierten Lendenwirbels. Als hauptsächliche Symptome traten auch hier Schmerzen

Abb. 7. In der Spitze des Processus spinosus des vierten Lumbalwirbels eine periostale Verdickung und ein getrennter Kalkschatten.

Abb. 8. Tuberositas deltoidea stärker als gewöhnlich hervortretend. Ihre Oberfläche uneben und ihre Struktur grob.

Druckempfindlichkeit und Schmerzen bei Muskelzug an der Insertionsstelle auf.

Röntgenuntersuchung. In den chronischen Fällen sind oft röntgenologische Veränderungen zu konstatieren. Als erste sichtbare Veränderung tritt kleinfleckige Atrophie ganz an der Oberfläche des Knochens bei der Insertionsstelle auf. Später wird die Kontur der Insertionsstelle uneben, und es sind darin in der Richtung der Sehne vorragende kleine Knochenzacken anzutreffen. Seltener zeigen sich in der Nachbarschaft der Insertionsstelle isolierte Verkalkungen. Der Kalk liegt augenscheinlich in der Sehne, wie überhaupt in den Fällen, von denen SANDSTRÖM den Namen Peritendinitis calcarea gebraucht. In unserem Material kommen einige solche Fälle (Abb. 1 und 2) vor, aber diese weisen immer auch Veränderungen in der Insertionsstelle selbst auf. Es liegt daher auf der Hand, dass zwischen Peritendinitis calcarea und den Schmerzen verursachenden Insertionsperiostitiden keine scharfe Grenze besteht. *In manchen Fällen sind also sowohl die Insertionsstelle am Knochen als das daselbst inserierende Ende der Sehne erkrankt. Mithin handelt es sich um eine Periosteotendinitis,*

die in chronischen Fällen oft zu röntgenologisch nachweisbaren Veränderungen an der Insertionsstelle führt.

Bei den Epikondylitiden des Humerus liefert die Röntgenuntersuchung meist ein negatives Bild. In einigen Fällen sind jedoch an der Oberfläche des Epikondylus Veränderungen der vorstehend beschriebenen Art zu sehen (Abb. 1).

Bei den Fersenschmerzen finden sich im Calcaneus oftmals periostale Veränderungen. Häufig beobachtet man in diesen Fällen am Tuber calcanei eine Sporenbildung, die offenbar schon vor der Erkrankung vorhanden gewesen ist. Die ersten Veränderungen im Röntgenbild sind denn auch in diesem Sporn anzutreffen (Abb. 2). Diese Veränderungen bestehen in kleinen atrophischen Flecken. In den schwersten Fällen breitet sich der Kalkschwund längs der Oberfläche des Calcaneus nach verschiedenen Richtungen aus (Abb. 3 und 4). In Abbildung 4 erstrecken sich die Veränderungen über ein sehr weites Gebiet.

Zum Vergleich haben wir die Röntgenbilder von 110 symptomfreien Fersen durchgesehen. Auf denselben zeigte sich in drei Fällen ein Calcaneussporn, aber er hatte in allen Fällen glatte und scharfe Grenzen und wies keine atrophischen Flecken auf. Dies beweist nur, dass die von uns gefundenen Veränderungen in den von uns beschriebenen Fersenfällen wirklich durch eine Fersenschmerzen verursachende Krankheit entstanden waren. Unter den von uns untersuchten symptomfreien Fersen fand sich an 10 eine kleine, aufwärts gerichtete Knochenzacke bei der Ansatzstelle der Achillessehne. Auch diese waren stets scharf begrenzt, und sonstige Veränderungen waren nicht festzustellen (Abb. 5).

Die Insertionsschmerzen können augenscheinlich in jeder beliebigen Insertion auftreten, der Calcaneus, der Epicondylus humeri und das Tuberculum majus humeri sind ihre Lieblingsstellen. Beispiele von selteneren Sitzen bieten die Abbildungen 6, 7 und 8.

In dem durch Abbildung 8 veranschaulichten Fall wurde eine histologische Probe entnommen. Im Knochen der Insertionsstelle findet man etwas mehr als in normaler Corticalis Höhlen, die mit lockerem Bindegewebe oder mit Knochenmark gefüllt sind. An der Insertionsstelle sieht man hier und da Häufchen von Entzündungszellen, hauptsächlich Lymphozyten. Die Bindegewebsfasern sind etwas mehr homogenisiert als gewöhnlich. Die Veränderungen sind also minimal.

Behandlung. Beinahe alle Autoren konstatieren, dass die Insertionsschmerzen schwer heilbare Affektionen sind. Schon VULLIET sagt, die Epikondylitis widerstrebe der Behandlung. FRANKE konstatiert 191[illegible], dass die gewöhnlichen antirheumatischen Behandlungsmittel nicht helfen. Die einzige empfehlenswerte konservative Therapie ist nach seiner Ansicht absolute oder relative Ruhe. Die Behandlungsdauer sei jedoch lang. Die erkrankte Stelle müsse mindestens vier Wochen immobilisiert werden.

bevor eine Besserung zu erwarten sei. In den schwersten Fällen empfiehlt FRANKE eine Wegmeisselung des Epicondylus als einziges erfolgreiches Behandlungsverfahren. Diese Methoden wurden dann bis zum Jahre 1926 angewandt, wo HOHMANN seine Operation beschrieb. Er machte einen kleinen Einschnitt in die Insertionssehne des Epicondylus lateralis und konnte dadurch die Insertionsstelle in vollständige Ruhe versetzen. 10 Tage nach der Immobilisation war die Operationswunde und zugleich die Krankheit geheilt. Einen ähnlichen Eingriff empfahl SPITZY bei schmerzendem Calcaneussporn. Diese operativen Verfahren sind bis in letzte Zeit als die besten Behandlungsmethoden angesehen worden.

Erst in allerjüngster Zeit ist Röntgenbehandlung bei Insertionsschmerzen in Gebrauch genommen worden. 1932 empfiehlt sie KAHLMETER, und RICHARZ teilt 1935 145 Fälle von »Periostalgien» mit, in denen er Röntgenbehandlung anwandte, und er bezeichnet sie in diesen Fällen als die Methode der Wahl. Er geht so weit, zu behaupten, dass die Diagnose in den Fällen, in denen die Röntgenbehandlung nicht hilft, falsch sei.

In allen unseren Fällen ist Röntgenbehandlung zur Anwendung gekommen. Die gegebene Bestrahlung war sog. mittelharte: Spannung 160 kV, Stromstärke 4—6 mA und als Filter 0.5 mm Kupfer und 1.0 mm Aluminium. Die Entfernung zwischen der behandelten Stelle und dem Fokus betrug 30 cm. Die Dosen wurden auf der Haut gemessen. Im allgemeinen wurden kleine einmalige Dosen von 100—150 r angewandt. Diese Dosis wurde mit Intervallen von einem oder ein paar Tagen 2 mal gegeben. Wenn die Beschwerden fortdauerten, wurde nach zwei Wochen eine gleichartige Behandlung administriert. Meistens wurde die Krankheit durch diese Therapie geheilt. Nur selten brauchte die Behandlung nach längeren Intervallen fortgesetzt zu werden. Gewöhnlich hatte sich der Zustand der Patienten schon nach der ersten Behandlung gebessert. Zuerst verschwanden die Schmerzen, und danach wurden die Bewegungen schmerzlos. Die Druckempfindlichkeit verschwand zuletzt. Das endgültige Resultat tritt erst 2—4 Wochen nach der Behandlung in Erscheinung, aber die Arbeitsfähigkeit kehrt oft schon nach einigen Tagen zurück.

Lokalisationen	Dauer	Geheilt	Gebessert	Rezidive	Nicht geheilt
Epicondylus humeri . . .	Unter 6 Mon. . . .	6	1	0	0
	6 Mon. od. mehr . .	3	0	0	0
Calcaneus	Unter 6 Mon. . . .	6	0	2	0
	6 Mon. od. mehr . .	7	1	0	1
Sonstige	Unter 6 Mon. . . .	1	1	0	0
	6 Mon. od. mehr . .	0	1	0	0
	Zusammen	23	4	2	1

Die Ergebnisse der nachträglichen Erkundigung, die $^1/_2$—6 Jahre nach der Behandlung stattgefunden hat, werden aus der vorgehenden Tabelle ersichtlich.

Bei der nachträglichen Erkundigung gingen Angaben über 28 Patienten ein. Von diesen sind 23 gesund geworden. Bei 10 Geheilten hatte die Krankheit bei Beginn der Röntgentherapie 6 Monate oder mehr gedauert. Von den Patienten sind 4 gebessert und zugleich arbeitsfähig geworden. Nur einer der Erkrankten teilt mit, dass die Behandlung nicht geholfen hatte. Nur in 2 Fällen ist ein Rezidiv eingetreten. Der eine von diesen war nach der Behandlung $3^1/_2$ Monate, der andere ein Jahr gesund.

Gegenüber den anderen Behandlungsmethoden hat die Röntgentherapie zahlreiche Vorteile. Sie ist erstens für den Patienten angenehm und ungefährlich. Sie kann poliklinisch gegeben werden, und oft kann der Erkrankte schon einige Tage nach der Einleitung der Behandlung mit seiner Arbeit beginnen. Die Immobilisations- und die chirurgische Behandlung, mit denen ebenfalls verhältnismässig gute Resultate erzielt werden, fordern viel mehr Zeit und in den meisten Fällen Anstaltsbehandlung.

Zusammenfassend stellen wir fest, dass *in den Ansatzstellen der Muskeln und Sehnen eine rheumathische Krankheit vorkommt, die in chronischen Fällen oft zu röntgenologisch nachweisbaren Veränderungen an der Insertionsstelle führt. Aus diesen Veränderungen kann man schliessen, dass die Insertionsstelle am Knochen und das sich daran anheftende Sehnenende erkrankt sind. Mit Röntgenbehandlung erzielt man bei dieser Krankheit ausserordentlich gute Resultate.*

ZUSAMMENFASSUNG

Die Insertionsschmerzen treten am häufigsten im lateralen Epicondylus des Humerus und im Calcaneus auf. Trauma und Überanstrengung sind die gewöhnlichsten auslösenden Ursachen dieser rheumatischen Krankheit. In chronischen Fällen führt diese Krankheit oft zu röntgenologisch nachweisbaren Veränderungen an der Insertionsstelle. Zuerst sind oberflächlicher Kalkschwund an der Insertionsstelle des Muskels oder der Sehne, später kleine periostale Knochenzacken in der Richtung der Sehne zu konstatieren. Mit Röntgentherapie werden bei dieser Krankheit ausserordentlich gute Resultate erzielt.

SUMMARY

Insertion pains occur most often in the lateral epicondyle of the humerus, and in the calcaneus. Trauma and overstrain are the most usual immediate causes of this rheumatic disease. In chronic cases, this disease often entails roentgenologically visible changes in the place of insertion. First of all, a superficial disappearance of calcium at the insertion of the muscle or the tendon can be noticed, later small periosteal bone thorns in the direction of the tendon. Exceedingly good results may be attained with roentgenotherapy in this disease.

RÉSUMÉ

Les douleurs des insertions musculaires apparaissent le plus souvent au niveau le l'épicondyle latéral de l'humérus et du calcanéum. Les traumatismes et le surmenage ont les causes les plus courantes de l'éclosion de cette affection rhumatismale. Dans es cas chroniques la maladie entraine des altérations visibles à la radiographie, dans la égion des insertions. On observe d'abord une rarefaction calcaire au niveau de l'inser- ion musculaire ou tendineuse, et, ensuite des petites épines osseuses périostales dans la irection du tendon. Les rayons roentgen donnent dans ces cas un excellent résultat hérapeutique.

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ROENTGENOLOGIC EXAMINATION OF ACUTE ABDOMINAL LESIONS[1]

by

J. Frimann-Dahl, M. D.

If one endeavours to peruse the literature on this great subject one meets with several authors who express their amazement that this method is still in so relatively infrequent use. When this has been the case it has certainly been caused partly by a certain fear of sending patients suffering from acute abdominal lesions for roentgen examination, and partly by the fact that the roentgen examination has often offered results of doubtful value.

A number of publications on this subject has been received from Sweden. LAURELL particularly, in numerous larger and smaller publications, has shown with singular lucidity and force that the roentgenologic examination may be of exceptionally great value in acute abdominal lesions. There further exist — to mention the chief ones — papers by ARNELL, BAGER, HULTÉN, GROTH, KNUTSSON, LILJA, GRÅBERGER, LUNDBERG, LINDBLOM, MOBERG, NYSTRÖM, PETRÉN, RENANDER, STENSTRÖM and WESTERBORN, dealing with subjects of various kinds within the roentgen diagnostics of acute abdominal lesions. Further, there are papers published by HELLMER and WULFF on the intravenous urography in cases of acute ureteral stones. From the other Scandinavian countries, particularly from Denmark, PANNER, NORDENTOFT, DE FINE LICHT and NØRREGAARD have published larger, more special papers on the roentgenologic examination in cases of intestinal occlusion. From Finland papers have appeared by GULLICHSEN on the same subject. From Norway there exist papers on intestinal obstruction by SEMB and HØYER, and by GREVSTAD more generally on acute abdominal conditions.

In the other European Literature we find papers from as far back as 1920. I would particularly mention KLOIBER's classical paper on the

[1] Introductory paper read at the Congress of the Northern Association of Medical Radiology held at Oslo in June 1939.

oentgen examination in intestinal obstruction. There are further papers n perforated ulcers by FINSTERBUSCH, GROSS and STOCKER, on abdominal emorrhage by ZUKSCHWERDT and KEMMLER, and more general papers y H. H. BERG and BADE. The Frenchmen COLANERI and LAQUIÈRE in a aper on roentgenologic examination of acute abdominal lesions very otly talk about *roentgenologie d'urgence*. In American literature in recent ears some more general papers have appeared, by STECHER, ABRAMS nd RENDICH, KELLY and DOWELL, PONEMON and HALL. All considered, may surely be said *that the Scandinavian contribution on this territory too little known universally*. It is a regrettable fact that in the ordinary xt-books on roentgenology and surgery this important subject has not en collected in larger survey chapters.

It is not possible in the time at disposal to give a more detailed reading n this subject. I have found it most suitable, therefore, to submit a naterial of my own.

Roentgen examination of acute abdominal lesions has not been performed systematically at the Roentgen Department of Ullevål Hospital until the last three years. I have had the opportunity myself of carrying out this work during the last year. The reason that I still submit a material comprising three years is to show the extent to which this method of examination is used at two large surgical departments with a total of 325 beds, intended also for emergency cases. I would like to avail myself of this opportunity of thanking the chiefs of the surgical departments ROSING BULL and CARL SEMB for their kindness in willingly placing the material at my disposal.

As the subject is actually of enormous extension only the largest and most important groups can come into question, and the review can only be schematical. In the course of the last three years the following cases have been examined:

Table 1

1.	Perforated ulcers	41
2.	Conditions of ileus	160
3.	Volvulus of the large intestine	13
4.	Ureteral stones	262
5.	Traumatic renal lesions	12
		488

In addition to these groups there are also a number of smaller ones, s acute pancreatitis, cholecystitis, paranephritis, intraperitoneal abscesses, intussusception, and so forth. These cannot be included here, however, as before mentioned.

Only a few remarks with regard to the roentgen anatomy before dealing with the separate groups. Perhaps in no other parts of the roentgen

diagnostics is it of such importance to know the normal variations in th roentgenogram as when it comes to the evaluation of acute abdomina lesions.

It is a well-known fact that the contours of the parenchymatous organ are visualized on the roentgenogram of the abdomen because of thei density and their sharp outline. As far as the kidneys are concerned th outlines appear more clearly due to surrounding fat. A linear clarificatio appears around the kidney and also near the psoas contours, which mus be supposed to be due besides to surrounding fat also to the Ott phenom enon. In the flanks we have several linear clarifications due to th same cause, located between the muscle layers. The most important however, is the clearing between the transverse abdominal muscle an the viscera, long since described as the extraperitoneal fatty layer. Just as important as the knowledge of the flank stripes is the familiarity wit the variegated multiplicity of normal variations of the form and size o the extraperitoneal fatty stripe from babyhood to advanced age, with filled and empty intestines, in lateral recumbency, erect position, and so forth, a fact already brought to attention by LAURELL.

Roentgen examination of *cadavers* with and without fluid or contrast substance in the abdominal cavity will give a more exact and detailed knowledge of these relations (fig. 1). This roentgenogram shows an abdomen from which the intestines have been removed, whereupon the parietal peritoneum has been smeared with barium mixture. It will be noticed that the peritoneum laterally in the flanks forms a fine, sharply defined brim or line, and that laterally to this there is a linear clarification 3—4 millimetres in width. By subsequent removal of the peritoneum it was very easy to convince oneself that this clearing was due to retroperitoneal fat. The modelling of the flank fat, the different widths of the flank stripes, et cetera, may be studied more closely in this manner.

It will lead too far, however, to discuss all the many normal variations. I would like to mention only that the fatty omentum may occasionally lodge between the intestines and the peritoneum and add to the width o the flank stripe, either on one side or on both sides (fig. 2). The omentum here is smeared with contrast and is observed lying evenly distributed t the flanks on both sides and down into the iliac fossae.

As a rule one may observe the flank stripe on the usual survey roentgenograms of the abdomen with a suitable exposure. When meteorism i present, which is practically always the case postmortally, the intestina air as in pronounced meteorism intra vitam laterally covers the extraperitoneal fatty layer which is chiefly developed in the dorsal half of th flanks. The meteoristic intestines may namely turn the *upper* lateral par of the abdominal wall outwards so that they cover the flank fat, whic is chiefly located *backwards*. It may be necessary, therefore, to take spe-

1.

2.

Fig. 1. The dense line is barium coating the parietal peritoneum. (The intestines have been removed — dissection.)

Fig. 2. The omentum has been smeared with contrast. It reaches out to the flanks and the parietal peritoneum (dissection).

cial roentgenograms of the flanks with the tube centred above the flanks or 15 degrees obliquely from the side. Then only is the extraperitoneal fat stripe distinctly visible.

If exudate appears in the abdomen, this usually first settles in the pelvis. Soon, however, it also moves outwards to the flanks and the parietal peritoneum. The medial border of the flank stripe becomes sharper and denser. This has been demonstrated in patients by LAURELL and HULTÉN, and in his many papers LAURELL has specially interested himself in this symptom. This may be beautifully proved directly by puncturing cadaver abdomens and at the same time injecting into free peritoneal cavity 400—500 c. c. water to which has been added a little barium. By varying the quantity of fluid injected into the abdomen all other signs of exudate are revealed, as has been described by clinical studies, such as accumulation of fluid in the pelvis, broad bands between the intestines, et cetera. These investigations have not yet been finished. I may state, however, that the method is very useful in order to learn the normal and the pathologic roentgen anatomy of the abdomen.

The technique which we use for examinations of patients suffering from acute abdominal lesions is mostly carried out according to the instructions furnished by LAURELL. In each separate case we are prepared for several possibilities, and the roentgenography and the fluoroscopic examination are adjusted according to the findings. Frequently a number of roent-

Fig. 3. Survey roentgenogram of the abdomen in left lateral recumbency. Air is seen in a niche on the side of the lesser curvature and air below the right dome of the diaphragm, as also in the right flank.

Fig. 4. Mechanical ileus of the small intestine photographed in supine posture with contrast in the small intestine. (The obstruction in the right iliac fossa.) At the same time is seen quite considerable colonic meteorism.

genograms are taken in erect or semi-seated position, in right and left lateral position, or special exposure of the pelvis after the patient has been seated for a while so as to collect the exudate in the declive parts. Recently we have tried to collect the exudate (particularly if it is small) in the flanks in lateral recumbent posture by placing the patient between two cushions. The exudate then runs forwards into the flank and may be examined with horizontal direction of the rays. This we carry out led by our experience with small exudates in the pleura which, it will be known, may be demonstrated in the same manner. The thorax is always examined fluoroscopically or roentgenologically if signs are observed of pathologic changes. A 4-valve apparatus supplies just as good as or even better exposures than a 6-valve apparatus. It is our opinion that medium hard rays, relatively short time of exposure and a thick Bucky diaphragm are preferable. The transport from the surgical departments should not be too long. The examination should not take too long a time. It is of importance, therefore, that physicians as well as nurses have experience with these examinations. Careful watch must be organised night and day.

Perforated Ulcers

My material here is comparatively small, and can hardly give anything rticularly new. 9 of the 41 cases examined revealed 9 negative roentgen ndings. The demonstration of free air in the peritoneal cavity naturally regarded as the most important symptom. Search is made just as care- lly, however, for other signs, in the first instance for signs of exudate d peritonitis. This more extensive examination has only been carried t for the last six months, however. These examinations were previously ten incomplete, the only question being of ascertaining whether or not ee air was present. In one case, in which the patient could only be amined in lateral recumbency, a long horizontal fluid level was found the abdomen, but no certain air cap. *It appears, therefore, as if in me cases one is able to observe a fluid level with no certain air*, presumably ue to the fact that the air cap is very thin.

In some cases from this last year it has been possible to demonstrate the ulcer directly with the aid of the gastric air in a niche on the side of the lesser curvature (fig. 3). In three cases it had to be regarded as probable that a duodenal ulcer was present due to deformity of the bulb, and this was subsequently verified by operation. *In many cases, therefore, the gastric air may be used as contrast medium for direct demonstration of the localization of the ulcer.* If there is perforation with free air in the peritoneal cavity, the stomach as a rule is slightly distended. In some cases, however, there may be very considerable distension in spite of large quantities of air below the diaphragm. *The quantity of gastric air, therefore, supplies no safe data as to whether or not perforation is present.* Barium contrast is not used as a rule. I am, however, inclined to support the views of STECHER and BADE when they state that when a couple of mouthfuls of contrast is used in a case of perforated ulcer no harm has been done, even if the contrast medium gets into free peritoneal cavity; this even makes it possible to make the diagnosis with certainty. It must be borne in mind that a couple of mouthfuls of contrast, as is now the routine in several acute abdominal lesions, is something else than a whole plateful of barium mixture, which was often used previously in conditions of intestinal obstruction, and then later was rather looked upon as contraindicated because of complications that occurred. The differential diagnosis as against acute pancreatitis may be difficult or impossible clinically, but then the examination of the patient with a couple of swallows of barium mixture may supply very safe data for the presence of a pancreatitis.

In a case of severe spontaneous pneumo-peritoneum a perforation was present of a sigmoiditis (diverticulum?). Further a case of a perforated

cholecystic gallbladder. In another case of acute pneumo-peritoneum from the Surgical Department III of Ullevål one was not able — all possible erroneous sources having been taken into consideration — to find and explanation of whence the air had entered into the peritoneal cavity.

Intestinal Obstruction

In order to give a record of the number of examined cases suffering from conditions of intestinal obstruction, I have compiled the material in tabular form:

Table 2

Distribution of 173 cases of ileus and volvulus

Period	Mechanical ileus	Paralytic ileus	Colonic volvulus	Total
1/5 36—1/5 37	28	13	5	46
» 37— » 38	50	6	7	63
» 38— » 39	49	14	1	64
Total	127	33	13	173

In all cases of clinically established cases of mechanical ileus except one, the roentgen examination revealed distended intestines with horizontal fluid levels. In the one case a high intestinal obstruction was present without distinct fluid level. The patient had not been examined with peroral contrast, however, in which case the stenosis would probably have been demonstrable.

As a rule the roentgen examination of a purely mechanical obstruction of the small intestine may be said to be comparatively easy. The signs of this are so familiar that I cannot go closer into this. The only thing I would like to mention is that the patient should not be given an enema previous to the examination, as this may cause an artificial fluid level to develop in the large intestine. Another important sign, which I would emphasize, is the lack of air in the colon. This has also been mentioned previously by several authors, for example ABRAMS, RENDICH and HØYER. This does not always hold good, however, as also expressly pointed out by ABRAMS and RENDICH, who state very correctly that the large gut is seldom air-filled in obstruction of the small gut. Of 75 own cases of obstruction of the small gut 39 cases showed absolutely no visible gas in the colon, 14 cases showed part gas-filling, and 22 cases showed small bubbles of air in some part or other of the colon.

We thus see that in approximately half of the cases of mechanical obstruction of the small intestine no air at all could be demonstrated in the large intestine. In approximately 20 per cent of the cases the air is easily visible in the large intestine, in approximately 30 per cent partly

Fig. 5. Mechanical ileus of the small intestine in standing posture. Large, garlandshaped coils, most in the upper part of the abdomen. In some smaller coils the fluid reaches different heights in the two segments.

Fig. 6. Same case with special exposure of the flank in dorsal recumbent posture. Characteristic rounded outline of the fluid-filled coils of small intestine, as also semicircular folds.

visible (generally as small bubbles of air). In one case we were able to make an exact local diagnosis of obstruction in the right iliac fossa from the appearance of the pre-stenotic coils (reversed U), in spite of the presence of slight gaseous distension of the cecum. In another case of mechanical obstruction of the small gut, which was examined several days after the onset of the attack, a couple of distended coils of small gut were observed in the medial part of the abdomen with fluid level in standing posture. No air in the colon, however. As there was a possibility of a high ileus, however, the patient was given a couple of mouthfuls of contrast, so that the small gut and the site of the obstruction was distinctly visible. Examination five hours afterwards showed that a considerable meteorism had developed in the rectum, the sigmoid and the descending colon (fig. 4).

Further I shall only show one single case of obstruction of the small intestine with large, broad, garlandshaped coils across the upper part of the abdomen (fig. 5). In this case a mechanical ileus was present with considerable fluid in the abdomen (mixture type). A special exposure of the flanks demonstrates (fig. 6) large, characteristic, fluidfilled coils with semi-circular folds.

Fig. 7. Peritonitis ileus, abscess with air bubbles in the appendiceal region. (Right lateral recumbency).

Often it is not possible to distinguish with certainty the mechanical ileus from the *paralytic*, as the bowel even if a true mechanical ileus is present, may be in a very quiet phase with small, hardly visible or audible movements. In other cases the movement or lack of movement of the fluid levels, their shape and extension as also signs of peritonitis, may allow a specific roentgenologic diagnosis. Here is seen a typical picture of a paralytic ileus with large, distended small guts, exudate in the abdomen and abscess in the right iliac fossa. Varying postures (right recumbent posture) demonstrates that the abscess air remains lying in the right iliac fossa (fig. 7) while the intestinal air rises towards the flank.

In spite of the roentgenologic signs available for differentiation between the mechanical and the paralytic ileus, there will always remain some doubtful cases. The roentgen examination, for example, may in some cases only supply information as to whether a condition of ileus is present, or that this is exceedingly probable. It is the clinician who must then in the first instance evaluate the findings and compare them with the clinical picture.

Obstruction of the large intestine. It is also as a rule fairly easy to demonstrate this condition. It must particularly be kept in mind that the distension of the gut must not necessarily appear just proximal to the site of the obstruction. In the presence of a stenosis of the sigmoid the distension will generally appear in the cecum. Gradually or by change of posture, gas will also accumulate immediately proximal to the stenosis. A distension of small gut, even with several fluid levels, may occur secondary to an insufficiency of the ileocecal valve.

The location of the obstruction has to be more exactly demonstrated with barium enema. Cases of obstruction of the large intestine have been admitted remarkably frequently on suspection of cancer. On the first examination it may often be difficult to make any other diagnosis than ileus with stenosis or stricture.

In this material the following figures are found:

Table 3

Obstruction of the large intestine, volvulus excepted

Gas and fluid levels in large intestine		Gas in small intestine			Cases of cancer of the colon
+	±	+	—	±	
28	1	12	15	2	23

Volvulus (of the colon) gives a typical and generally easily interpreted roentgenologic finding. It is diagnosed on survey exposures made in different recumbent postures, with or without barium enema. On subsequent perusal of the material (13 cases) I have only been able to confirm a number of the signs described by LAURELL in such an admirable way in his great paper on volvulus.

Ureteral stones with acute symptoms do not actually belong among abdominal lesions. Differential diagnostically, however, this is such an important and large group, that it has got to be included. The most important point, as previously emphasized by WULFF and HELLMER, is that the patient is examined with intravenous urography during attacks immediately on admission into the surgical departments. It then appears that practically all the cases of ureteral stones give positive roentgen findings. In co-operation with ARNESEN I have classified our cases in the following table:

Table 4

Admitted under the diagnosis:

	Total	Acute abdomen	Stones of the urinary tract	Ileus	Acute appendicitis	Perforated ulcer	Cholelithiasis
		Abdominal colic	Nephrolithiasis?				
1936 . . .	67	24	30	—	11	1	1
1937 . . .	81	23	47	3	7	1	—
1938 . . .	114	33	56	2	20	1	2
	262	80	133	5	38	3	3

262 cases of ureteral stones examined with intravenous urography during attacks immediately after admission.

This table shows how important the *immediate urography* has become for the differential diagnosis in the motley multiplicity of lesions represented by »acute abdomen».

Finally only a few words regarding the roentgen examination in *abdominal traumatism*. After LAURELL's last paper on this subject a new chapter has opened in the diagnostics on the abdominal lesions. At Ullevål Hospital we are constantly admitting patients suffering from these lesions, and the majority of them are now examined roentgenologically. I shall here only mention the largest group in this material, the kidney lesions, with which we have relatively great experience.

Acute kidneys lesions (*ruptures*) are examined with intravenous urography immediately on admission. This examination has proved to be quite harmless to the patients and to be of very great value in the diagnosis. 12 cases in all have been examined in the course of the last 3 years. In cases of renal trauma and subcutaneous abdominal lesions it is not only a question of demonstrating whether or not the kidney has been compromised, but to demonstrate blood in the pelvis and in the abdomen, peritoneal irritation, basal atelectases in the lungs, and so forth. In short, almost the entire collected experience at disposal with regard to the roentgenology of acute abdominal lesions comes into use here. This material will later be subject to a separate publication. For the sake of brevity the findings have been compiled schematically.

Roentgenologic Symptoms in 12 Cases of Renal Lesions examined with Intravenous Urography

A.	*Survey roentgenogram before contrast:*	
	Hematoma	9 patients
	Scoliosis	8 »
	Meteorism	6 »
	Enlarged kidney	2 »
	Intraperitoneal exudate	2 »
B.	*Intravenous Urography:*	
	Deformity of the renal pelvis	8 patients
	Reduced excretion	5 »
	Contrast outside the renal pelvis	3 »
	Dilatation of renal pelvis and ureter	1 »

Conclusion

Roentgenologic examination of acute abdominal lesions is a very important and comprehensive examination, a speciality within the speciality of roentgen diagnostics. One should not confine oneself to separate examinations or separate lesions, thus making limited groups, but one should collect experience by examining roentgenologically as many cases as possible. This work should be organised in the hospital department with expert help at disposal night and day. The roentgen department should lie close to the surgical departments, with easy transport facilities. Intimate co-operation with the surgeons is an absolute necessity.

SUMMMARY

After a brief introductory review of the literature on the roentgen examination in acute abdominal lesions, attention is drawn to the fact that the Scandinavian contribution on this subject appears to be too little known. A schematical record is then made of the most important groups of acute abdominal lesions, and the roentgen examination of these as carried out at the Oslo Municipal Hospital Ullevål during the last 3 years. This examination must be regarded as very important. Lengthy experience is necessary as well as a special organisation of the correct running of the examination. Intimate co-operation with the surgical departments is necessary.

ZUSAMMENFASSUNG

Nach einem kurzen einleitenden Bericht über die Literatur bezüglich der Röntgenuntersuchung akuter abdominaler Läsionen macht Verf. auf die Tatsache aufmerksam, dass der skandinavische Beitrag zu diesem Gegenstand zu wenig bekannt ist. Er gibt dann ein schematischer Verzeichnis der wichtigsten Gruppen von akuten Abdomenläsionen und der an ihnen in den letzten 3 Jahren im Osloer Munizipalkrankenhause in Ullevål vorgenommenen Röntgenuntersuchungen. Diese Untersuchung ist als sehr wichtig zu betrachten. Es bedarf langer Erfahrung wie auch besonderer Organisation der korrekten Durchführung der Untersuchung. Ferner ist enge Zusammenarbeit mit den chirurgischen Abteilungen unbedingt erforderlich.

RÉSUMÉ

Après une courte introduction sur la littérature des examens radiologiques dans les lésions abdominales aiguës, l'attention est attirée sur le fait que la contribution scandinave à ce sujet paraît être trop peu connue. Une exposé schématique vient ensuite des plus importants groupes de lésions abdominales et de l'examen radiologique de ceux-ci tel qu'il a été pratiqué à l'Hôpital municipal d'Oslo Ullevål pendant ces 3 dernières années. Cet examen doit être considéré comme très important. Une longue expérience est nécessaire ainsi qu'un organisation spéciale du cours de l'examen. Une intime coopération avec le service chirurgical est nécessaire.

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DEGLUTITION ANOMALY SIMULATING HYPOPHARYNGEAL CANCER[1]

by

Sölve Welin

In the course of the past 3 years I have in 5 patients come across a deviation from the normal act of swallowing which I have not found described in the literature and which therefore may merit to be recorded as a contribution to the patho-physiology of deglutition especially as it has been of decisive importance for the clinical interpretation of the cases.

The first of the five cases was that of a business man, aged 42, who for some time had had difficulty in swallowing solid food and been complaining of a well marked sensation of a foreign body in the throat. The patient at first consulted a general practitioner who under the diagnosis of oesophageal cancer referred him to an ear, nose and throat specialist. From here the patient was sent to us for roentgen examination.

The second case was that of a woman, aged 52, who besides certain difficulties in swallowing mostly complained of fits of coughing at and after meals.

The 3 remaining patients were also women over 50 years of age; 2 of these had only had their symptoms for shorter periods of time, 3 weeks and 2 months respectively, while the 3rd patient had a history of no less than 25 years. No wonder she had had plenty of time to consult a number of doctors in Sweden as well as abroad and through diligent study of popular medical writings she gradually developed well marked cancer phobia although no examiner, whether general practitioner or specialist, had ever been able to find any fault with the patient. Still her difficulties with swallowing and inability to take anything but minced food persisted and she was now going to a specialist for repeated control examinations. It was at one of these that I was asked to examine her under roentgen.

[1] Submitted for publication, July 24, 1939.

The classical description of the act of deglutition dates from MAGENDIE in 1817. Since that time the study of deglutition has been greatly facilitated by the introduction of roentgenography, and by fluoroscopy we find its well known stages appearing as follows. To begin with the opaque medium is observed in the anterior part of the mouth whence it is carried backwards by chewing and lingual movements to the base of the tongue and pharynx. When come so far voluntary movements cease and the swallowing reflex is called into action. The first thing now seen is that the larynx and the hyoid bone are lifted upwards and also to some extent forwards. At the same time 3 out of the 4 possible routes for the food become closed, viz. 1) the way back to the mouth by the anterior pillars of the fauces becoming tense and approaching one another, 2) the passage to the nasopharynx by the formation of a transverse ridge on the posterior and lateral walls of the pharynx which ridge becomes pressed against the posterior pillars, these having simultaneously approached one another and 3) the passage to the larynx by closure of its entrance.

The mechanism involved in the closure of the larynx has been the centre of great interest and the views held about it have varied considerably. As early as 1850 it was maintained by JOSEPH HYRTL in his textbook that admittance to the larynx during the act of swallowing was prevented by the epiglottis acting as a trap-door over the ostium laryngis and in so doing served as a bridge for the food. In 1892 ANDERSON-STUART put forward his view that the entry to the larynx was closed without aid of the epiglottis which during the whole act of deglutition would maintain its upright position and the food thus glide down over the laryngeal surface of the epiglottis. In 1911 however M. SCHEIER was able to show with great clearness that the epiglottis bends right down, putting itself as a proper lid over the introitus laryngis, causing the food to glide down over its lingual surface. Mots workers on this subject after SCHEIER have arrived at the same wiew.

Thus in a paper of 1931 on the normal act of deglutition LAURELL showed in a sketch that the epiglottis at first occupies a vertical position during deglutition, later to take up roughly a horizontal position. Yet there are still many who maintain that the epiglottis during the whole act of swallowing retains its upright position. Among these authors we find BARCLAY, an English roentgenologist, who published his work on the normal act of deglutition as late as 1932. Further, NEGUS has arrived at the same result and in his book »Våra livsförrättningar» THUNBERG says that the current view that the closure of the pharynx takes place by the epiglottis being brought down over the laryngeal opening is no longer tenable. THUNBERG has arrived at this view since the larynx is protected even in persons in whom the epiglottis is missing.

It is clear therefore that the opinions held of the behaviour of the

epiglottis at and its importance for the act of swallowing are much at variance. It has indeed been found that closure of the entrance of the larynx also depends upon several other factors, such as rising of the larynx, diminution of the aditus and above all closure of the glottis.

Thanks to the great technical strides made by radiography in recent years there is now no longer any difficulty in following the act of deglutition. In spite of the great rapidity with which it takes place it is thus

Fig. 1. The position of the epiglottis before swallowing.

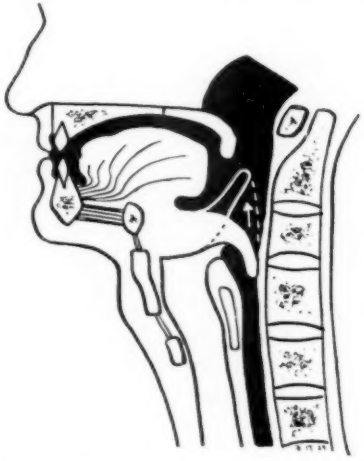

Fig. 2. The position of the epiglottis during the latter part of the act of deglutition.

quite easy to observe how the epiglottis from its upright position (fig. 1) bends down, whereby its lingual surface comes in contact with the contrast medium (fig. 2). This bending takes place as soon as the contrast has come in contact with the epiglottis, this then retaining its position till all the contrast has passed by. It then resumes its original position by way of a rapid turning back motion which does not seem to be met with any resistance.

In the 5 cases, on the other hand, related above the course of action was quite a different one in so far that the return motion only took place with great difficulty. It could thus be seen under the screen how the lingual surface of the epiglottis exceedingly slowly resumed its upright position. It took place in a jerky manner. After having finished gliding against the posterior pharyngeal wall, and the resistance having thus ceased, the epiglottis rose with quite a jerk. During the upward movement the epiglottis became more angularly bent than normal while the sagittal diameter of the pharynx at the same time seemed smaller than usual.

Since neither the roentgen examination nor hypopharyngoscopy had shown the epiglottis to be longer than normal the cause of the trouble might be found in some nervous disturbance with the result that the larynx is either not drawn sufficiently far forwards during the act of deglutition or else resumes its initial position too quickly.

Finally I would like to point out that in these cases there was no abnormal contrast filling of valleculae epiglotticæ or sinus piriformes but that it was the direct observation alone which led to the diagnosis.

By way of commentary the whole sequence of events may be summarised as follows. These 5 patients who had all reached the cancer age complained of so well marked a sensation of foreign body in the throat and of difficulty in swallowing that clinicians suspected cancer of the hypopharynx or oesphagus. Radiography however showed no signs of malignant infiltration, nor did the subsequent hypopharyngoscopy and oesophagoscopy lend any support to the clinical suspicion of malignant new growth. Nor has the clinical or roentgenological examination given any indications of PLUMMER-WINSON's disease. Nor can there be any question of bulbar paralysis since in this condition as well, as myasthenia gravis, poliomyelitis and botulism we get a generalised muscular paresis. The only explanation that remains is therefore that kinetic sort of dysphagia, which I have observed and which I have not found in any other but these 5 patients, in spite of a series of controls numbering several hundreds of cases. Whether then this dysphagia has its origin from local changes in the epiglottis, as e. g. diminished elasticity in the epiglottis. or in some neuromuscular affection along the atactic line of disturbed coordination, might still remain an open question.

SUMMARY

The author describes a deglutition anomaly consisting in a hampered return of the epiglottis normally bent down during the act of swallowing. The 5 patients in whom this dyskinetic dysphagia was observed all showed symptoms of hypopharyngeal cancer.

ZUSAMMENFASSUNG

Verf. beschreibt eine Schlinganomalie, die darin besteht, dass die Rückkehr der während des Schluckaktes normal nach unten gebogenen Epiglottis gehemmt ist. Die 5 Patienten, bei welchen diese dyskinetische Dysphagie beobachtet wurde, zeigten alle Symptome von hypopharyngealem Kanzer.

RÉSUMÉ

L'auteur décrit une anomalie de la déglutition, consistant en un obstacle au mouvement de l'épiglotte qui normalement doit s'abaisser durant la déglutition. Les 5 sujets chez lesquels cette dysphagie dyskinétique fut observée ont tous présenté les symptômes du cancer hypopharyngien.

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AUS DEM RÖNTGENINSTITUT DER UNIVERSITÄT ZÜRICH. PROF. DR. H. R. SCHINZ

ZUR FRAGE DER EMBOLIE BEI HYSTEROSALPINGOGRAPHIE MIT LIPIODOL[1]

von

O. Walther. Assistent.

Seit Le Laurier 1913 und Gary 1914 die ersten röntgenologischen Darstellungen des Uterus und der Tuben gelangen, wurde intensiv weiter an dieser Methode gearbeitet. Das anfangs verwendete schädliche Collargol wurde bald durch das Lipiodol und das Jodipin ersetzt. Aber bald wurden auch mit diesen Kontrastmitteln Misserfolge bekannt, zuerst mit dem Jodipin, später auch mit dem Lipiodol. Es handelte sich vorwiegend um das Aufflackern von alten entzündlichen Prozessen in den Tuben und dem Uterus, das Auftreten von Sepsis, selten um Jodismus und Embolisierung der Uterus- und Beckenvenen. Diese Arbeit hat die Aufgabe, das Eindringen des Lipiodols in das Venensystem und die daraus entstehende Fettembolie experimentell zu studieren, wobei uns vor allem die organischen Veränderungen und die Ausscheidung des Kontrastmittels interessieren.

In der uns zugänglichen Literatur finden sich viele Abhandlungen, die über die Hysterosalpingographie, deren gute Resultate und oft aber auch über die verschiedenen Gefahren berichten. Wie schon erwähnt wird nur die Embolie durch das Kontrastmittel behandelt, während die übrigen Komplikationen und Schädigungen der Methode weggelassen werden, da dieselben schon eingehend behandelt wurden.

Bis jetzt sind 18 Fälle bekannt geworden, die meistens einzeln publiziert wurden. Klinisch war in 9 Fällen nichts Bemerkenswertes oder Typisches für eine Embolie nachzuweisen. 5 Mal fand sich ein langdauernder Hustenreiz, wovon drei mal mit blutigem Sputum.

Einmal wurde ein kurzdauernder heftiger Schmerz im Unterbauch beschrieben, der vom Autor als Zeichen des Eintrittes des Lipiodols in

[1] Bei der Red. am 8. VII. 1939 eingegangen.

die Uterusvenen angesehen wird. Bei fünf Frauen bestanden die klassischen Zeichen der Fettembolie, wie langdauernder Schwindel, temporäre Amaurose, Gedächtnisschwäche, Hustenreiz mit blutigem Sputum und positiver Eiweissbefund im Urin.

Röntgenologisch sind die Befunde selten, aber wenn beobachtet ungefähr gleich. Sie sind nur während kurzer Zeit nachzuweisen und deshalb oft übersehen worden. Es fanden sich, entsprechend dem Verlauf der Beckenvenen, besonders der Vena ovarica, Vena iliaca communis und Vena cava inferior, in acht Fällen bandförmige homogene Streifen, die meist nach kurzer Zeit (zehn bis sechzig Minuten) nicht mehr nachzuweisen waren. CARAVEN konnte diese röntgenologischen Befunde am Operationspräparat bei einer Totalexstirpation des Uterus und der Ovarien histologisch bestätigen, während PUYOL und VANRELL an Leichenversuchen genau die gleichen Bilder erzielen konnten. HEMMELER, EICHLER und LIN fanden fleckige Lungenverschattung und zweimal typische Lungeninfarkte, die nach einigen Wochen nicht mehr nachzuweisen waren. Klinisch bestand dabei länger dauernder Hustenreiz, vereinzelt blutiges Sputum und Stechen auf der Brust. In einem einzigen Fall trat kurz nach dem Eingriff der Tod ein. Autoptisch konnte die Eintrittstelle des Kontrastmittels in einer grossen, stark erweiterten Vene eines submukös gelegenen Myomknotens gefunden werden. Dieselbe war durch das Instrument verletzt worden. Es handelt sich also in diesem einzigen Falle um einen ungünstigen anatomischen Nebenbefund. Eine ebenfalls tödlich verlaufende Embolie ist bekannt nach einer Urethrographie bei Striktur, wo ebenfalls die Eintrittstelle in einer verletzten und dadurch eröffneten Vene der Urethralschleimhaut gefunden wurde. Ausser den erwähnten schweren Fällen sind die beschriebenen Komplikationen ohne nachweisbaren Schaden nach Tagen oder Wochen abgeheilt.

Nach BÉCLÈRE ist die Gefässinjektion zu erkennen an einer Verschattung der Uteruswand und an breiten Streifen längs der Wirbelsäule. Diese beiden röntgenologisch nachweisbaren Zeichen sind aber nur für kurze Zeit sichtbar, da der Blutstrom das Lipiodol rasch abtransportiert. Nach DALSCASE und LIN entstehen die Embolien bei einem Injektionsdruck von über 200 mm Quecksilber, bei allgemeiner Narkose, bei infantilem Uterus und kurze Zeit nach der Periode. Ferner soll in einzelnen Fällen eine pathologische Durchlässigkeit der Gefässe und Läsionen der Uterusschleimhaut, hervorgerufen durch Instrumente eine gewisse Rolle spielen. Nach PUYOL sind kleine Embolien der Beckenvenen sehr häufig, klinisch völlig ungefährlich und dem venösen Reflux bei der retrograden Pyelographie gleichzusetzen. Die beiden erwähnten Todesfälle sind auf Verletzungen der Schleimhäute durch Instrumente zurückzuführen. Bei genügenden Vorsichtsmassnahmen beim Injizieren

Abb. 1. Kaninchen Nr. 1056. Aufnahme direkt nach der Injektion von 1 ccm 40 %igem Lipiodol in die re. Ohrvene.

Abb. 2. Dasselbe Kaninchen. 7 Monate nach der Injektion von 1 ccm 40 %igem Lipiodol in die re. Ohrvene. Histologisch noch zahlreiche Lipiodoltropfen in den kleinen Lungenarterien.

des Kontrastmittels sind schwerere Komplikationen sicherlich zu vermeiden.

Wie schon erwähnt sind die röntgenologisch sichtbaren Zeichen einer Gefässfüllung durch Lipiodol nur für kurze Zeit nachweisbar. Es fragt sich nun, wohin das Kontrastmittel durch den Blutstrom gelangt, wo es stecken bleibt, welche Veränderungen es in einzelnen Organen erfährt, welche Schädigungen im Organismus entstehen und welches die Art und der Ort der Ausscheidung ist. Wir haben nun experimentell versucht, diese verschiedenen Fragen zu beantworten. Wir injizieren 1 cm^3 40 %iges erwärmtes Lipiodol langsam in die Ohrvene eines Kaninchens, um eine mittelschwere Fettembolie zu erzeugen, welche das Tier nicht zu sehr schädigt, um während Wochen die oben erwähnten Fragen studieren zu können. Klinisch konnte bei dieser Menge ausser einer kurzen leichten Dyspnoe nichts festgestellt werden. 15 und 30 Minuten nach der Injektion wurden Röntgenaufnahmen des ganzen Kaninchens gemacht, eine weitere kurz vor der Tötung und eine letzte der sezierten Organe.

Auf den Röntgenbildern des Thorax findet man anschliessend an die Injektion die grössten Lungenarterien gefüllt mit dem Kontrastmittel. Im Verlauf von Stunden werden die homogenen, dichten, und verzweigten

Abb. 3. Kaninchen Nr. 1033. Aufnahme sofort nach der Injektion von 1 ccm Lipiodol in die re. Ohrvene.

Abb. 4. Dasselbe Kaninchen. Lungen und Herz 30 Stunden nach Versuchsbeginn.

Schatten kleiner und feiner, um später vollständig zu verschwinden. Das Lipiodol wird durch die Herzkraft allmählich in die kleinsten Arterien und Kapillaren hineingepresst, wo es langsam durch Verseifung und zelluläre Resorption verschwindet, weshalb es dann nicht mehr röntgenologisch nachgewiesen werden kann (s. Abb. 1 u. 2). Auf den Aufnahmen der obduzierten Lungen ist das Kontrastmittel bei kurzer Beobachtungszeit in Form vieler feiner aneinandergereihter schwarzer Punkte sichtbar (s. Abb. 3 u. 4), während nach Monaten nur noch vereinzelte sichtbar sind. Die Tiere wurden alle durch Genickschlag getötet. Die Obduktion ergibt immer den gleichen Befund, quantitativ nach der Überlebensdauer verschieden. In den Pleurahöhlen fand sich stets ein klares Transsudat von verschiedener Menge. Nur im Probeversuch, wo das Tier kurze Zeit nach Versuchsbeginn ad exitum kam, da wir 4 ccm Lipiodol injizierten, fand sich ein haemorrhagisches Transsudat und eine deutliche Dilatatio cordis. Die letztere war aber sonst nie nachzuweisen. An der Pleura waren stets viele punktförmige Blutungen vorhanden, vereinzelt auch am Endokard. Lungenödem bestand nicht. Sonst zeigten die Organe makroskopisch nichts Abnormes. Wir möchten an dieser

Stelle Herrn Prof. Dr. CLAIRMONT danken für die Überlassung des Laboratoriums zur Anfertigung der histologischen Schnitte.

Durch die Einspritzung von Lipiodol in eine Vene entsteht im Tierexperiment eine Fettembolie. Die örtlichen Veränderungen der einzelnen Organe, in unserem Falle hauptsächlich der Lungen, hängen ab von der Menge des eingespritzten Kontrastmittels. Dieses liegt in den mittleren und kleinen Arterien, selten in den Kapillaren, was durch die hohe Viskosität bedingt ist, da im Tierexperiment die Herzkraft nicht vermindert ist. Die Gefässe sind erweitert und hyperämisch, die elastischen Fasern, die Endothelien und die Adentitiazellen der Gefässwände sind intakt. Lungenödem konnte nur im Probeversuch nachgewiesen werden. Die von WEGELIN und KOYO beschriebenen Atelektasen und sekundären Wucherungen des Alveolarepithels konnten nicht beobachtet werden. Vereinzelt finden sich in beieinander liegenden Alveolen kleine Blutaustritte, mit vielen Erythrozyten und einzelnen desquamierten Epithelien, die hier und da Lipiodol und Haemosiderin enthalten. Die Septen sind stets intakt. Typische haemorrhagische Infarkte wie sie vor allem LIN beschrieben hat, konnten nicht nachgewiesen werden. Die Ursache liegt in der Versuchsanlage, da wir nur kleine Mengen injizierten. Entzündliche Herde in der Umgebung der Lipiodoltropfen fanden sich nie, wie sie E. WAGNER und DIETRICH als Folge von mitverschleppten Bakterien beschrieben haben. Bei der Hysterosalpingographie ist dies sehr möglich wie die Fälle von HEMMELER und EICHLER beweisen. In einzelnen grossen interlobären Septen in der Nähe der Bronchien finden sich kleine Lymphozyteninfiltrate. In unsern Versuchen haben nur minimale Mengen des Kontrastmittels das Lungenfilter passiert, weshalb in den übrigen Organen des grossen Kreislaufes nur vereinzelte Lipiodoltropfen nachzuweisen sind, die auch mikroskopisch keine Veränderungen hervorrufen konnten. Ist aber die in die Venen eingedrungene Menge eine grosse, so sind organische Veränderungen in der Lunge und im grossen Kreislauf und sogar ein tödlicher Ausgang möglich. Die primäre Todesursache bei Fettembolie ist das Erlahmen des Herzens. Nach KOYO ist der rechte Ventrikel stark dilatiert, mit Blut gefüllt, während der linke Ventrikel fast völlig leer ist. Die Blutdruckkurven ergeben zuerst eine starke Senkung, dann einen kurzen Wiederanstieg und schliesslich eine rasch zunehmende Senkung bis zum Tode. Die Ursache ist die plötzliche Verstopfung einer grossen Zahl von Lungenarterien. Nach DIETRICH tritt der Tod erst ein, wenn zwei Drittel der Lungengefässe verstopft sind.

KERBER fand bei dosierter Luftembolie an Kaninchen autoptisch röntgenkymographisch unter Anwendung der Kontrastblut-Methode und elektrocardiographisch reichlich Luftblasen in den Koronarvenen, welche eine Abflusshinderung und Stauung hervorrufen, der eine rasch einsetzende und fortschreitende Koronainsuffizienz folgt. Der Autor

glaubt, dass dies die primäre Todesursache bei Luftembolie sei. CLAIRMONT hat schon 1910 auf das Vorhandensein von Luftblasen in den Koronargefässen bei Luftaspiration hingewiesen. Wir konnten bei unsern Versuchen mit Lipiodol nie etwas davon in den Koronargefässen nachweisen.

Übersteht ein Versuchstier die Embolie, so spielen sich am liegengebliebenen Fett oder Lipiodol bestimmte Vorgänge ab, die zu dessen Entfernung dienen. Die heutige Auffassung der Veränderung des Fettes oder Öles ist folgende: Der wichtigste Vorgang ist die Verseifung an Ort und Stelle und erst in zweiter Linie kommt die sicherlich nicht zu vernachlässigende zelluläre Resorption. BENNECKE hat die komplizierten Vorgänge pathologisch-anatomisch untersucht. Seine Auffassung ist heute die massgebende. An den Emboli beobachtet man eine Zerklüftung in einzelne kleine Teile and anschliessend die völlige Zersplitterung des Tropfens. Der Grad und die Ablaufgeschwindigkeit dieser Erscheinung hängt ab vom Gehalt von Fettsäuren und der Verwandschaft des Fettes, d. h. arteigenes Fett wird besser und schneller gespalten. Nach BENNECKE bringt das Blutserum, das Lipasen enthält, die Oberfläche des Fettropfens zur Verseifung. Nach dem Gad'schen Versuch tritt sofort nach Berührung von Fettropfen und Sodalösung Seifenbildung auf mit nachfolgender Zertrümmerung des Tropfens. Dasselbe ist zu beobachten, wenn Fett mit Blut in Berührung kommt. Diese Emulsionserscheinungen spielen sich vorwiegend an den Enden der Tropfen ab, weshalb an einen gröbern Embolus zahlreiche kleinere Tropfen angelagert sind und als dessen Abkömmlinge sich in die anschliessenden Kapillaren vorschieben (Abb. 5.) In dieser Art und Weise erfolgt die Zersplitterung und Auflösung des Tropfens. Nach WEGELIN ist dieser Vorgang zu vergleichen mit der Verdauungslipämie, bedingt durch die Serumlipase, die nach WOHLGEMUTH in allen Organen, besonders aber in Leber und Lungen vorhanden ist.

ROGER, BINET, SICARD, FORESTIER und besonders FABRE in Paris haben die chemischen Abbauvorgänge und besonders die Ausscheidung des Lipiodols experimentell an Hunden geprüft. Sie fanden, dass gewöhnliches Fett durch die hohe lipolytische Funktion des Lungengewebes rasch abgebaut wird, da nach ausgiebiger Fettnahrung im Blut des linken Ventrikels der Fettspiegel bedeutend niedriger ist als rechts. Das Blut erhält in den Lungen eine vermehrte lipolytische Funktion, die nach den Autoren unabhängig ist von der Oxydation. R. FABRE hat die besondern Verhältnisse des Lipiodols an Hunden chemisch untersucht. Er fand dass, entgegengesetzt der Meinung Vieler, das Lipiodol im Körper durch die Lipasen in seine Hauptbestandteile Öl und Jod gespalten wird und zwar im Unterhautzellgewebe bedeutend langsamer als in der Muskulatur und im Epiduralraum rascher als im Rückenmarks-

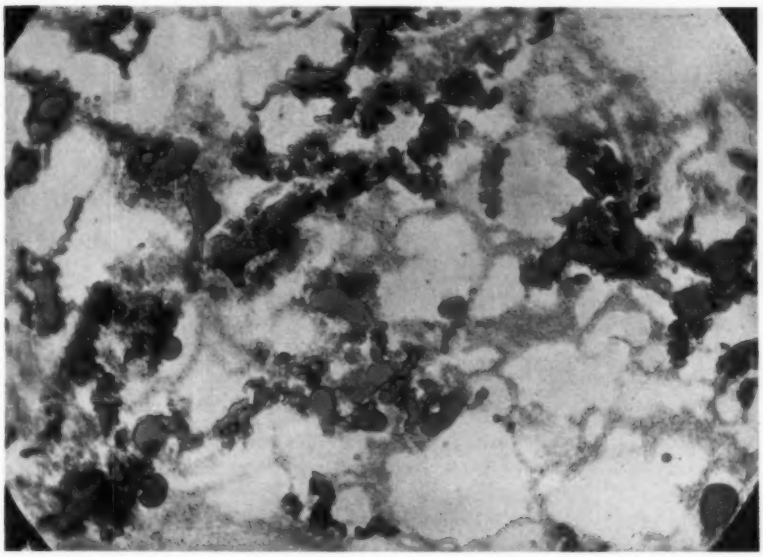

Abb. 5. Kaninchen Nr. 1050. Injektion von 1 ccm 40 %igem Lipiodol in die re. Ohrvene. Lunge. Fettfärbung. Vergrösserung 80 ×. 12 Stunden nach Versuchsbeginn.

kanal. Die intensivste und rascheste Spaltung tritt ein im Darm und im Gefässystem. Das Jod wird als Jodkali vorwiegend durch die Nieren ausgeschieden, in geringen Mengen durch Schweiss- und Speicheldrüsen im Darm. Bei der intravenösen Injektion von Lipiodol tritt ein massiver Abbau in den ersten sechs bis acht Tagen ein, dann nehmen die ausgeschiedenen Jodmengen im Urin rasch ab bis auf einige wenige Milligramm pro Tag, die noch während Wochen nachzuweisen sind. FABRE hat die Jodbestimmung im Urin nach der Methode von BERNIER und PIÉRON durchgeführt. Die Tagesmengen sind sehr klein. Der Zeitablauf und die ausgeschiedenen Jodmengen im Urin bei verschiedener

Intravenöse Injection von 2 ccm Lipiodol.

		I.	II.			I.	II.			I.	II.
		gr.	gr.			gr.	gr.			gr.	gr.
1. Tag	Jodgehalt im Tagesurin.	0.0190	0.0080	8. Tag	Jodgehalt im Tagesurin.	**0.0560**	0.0288	15. Tag	Jodgehalt im Tagesurin.	0.0052	0.0092
2. »		0.0326	0.0087	9. »		0.0396	0.0240	16. »		0.0050	0.0070
3. »		0.0346	0.0136	10. »		0.0310	0.0205	17. »		0.0046	0.0048
4. »		0.0386	0.0185	11. »		0.0190	0.0182	18. »		0.0031	0.0032
5. »		0.0431	0.0215	12. »		0.0142	0.0153	19. »		0.0020	
6. »		0.0438	0.0263	13. »		0.0087	0.0127	20. »		0.0019	
7. »		0.0461	**0.0296**	14. »		0.0060	0.0104				

Jodbestimmungen im Urin bei Hunden nach intravenöser Injektion von 2 ccm Liodol. Nach R. FABRE, Paris.

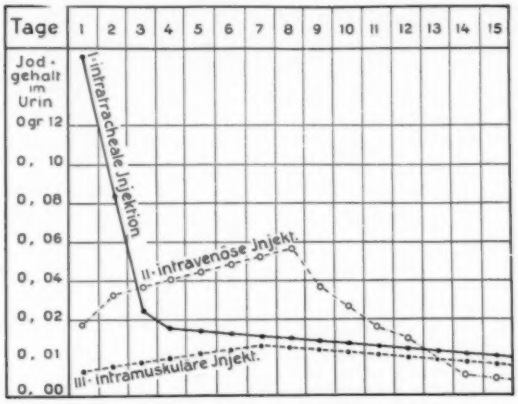

Abb. 6. Verschiedenheit der Jodausscheidung im Urin bei Hunden bei verschiedener Applikation des Lipiodols nach R. Fabre. Paris.

Anwendung des Lipiodols im Tierexperiment sind am besten illustriert durch das Diagramm. Fälle von Jodschädigungen sind bei der Hysterosalpingographie nicht beobachtet worden trotzdem sie bei diesem raschen Abbau des Kontrastmittels bei Embolisierung zu erwarten wären.

Wir haben nun bei unsern Versuchen mit Kaninchen ebenfalls die ausgeschiedenen Jodmengen chemisch gemessen, leider aber ohne Erfolg, da für diese Reaktion speziell eingeübtes Personal vorhanden sein muss, was bei uns nicht der Fall war. Wir haben uns deshalb erlaubt

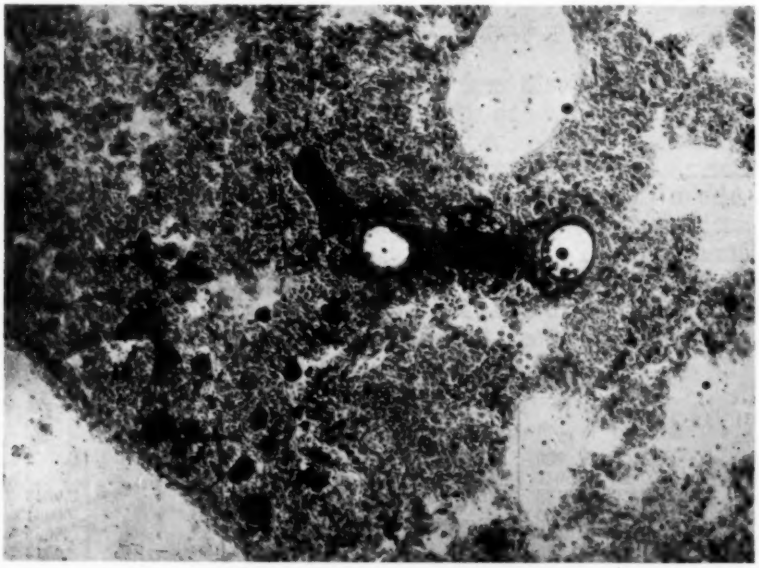

Abb. 7. Kaninchen Nr. 1056. Injektion von 1 ccm 40 %igem Lipiodol in die re. Ohrvene 7 Monate nach Versuchsbeginn. Lunge. Fettfärbung. Vergrösserung 120 ×. Endothelwucherung mit Lipiodoltropfen.

der Vollständigkeit halber die Resultate von R. FABRE wiederzugeben.

Neben dem chemischen Abbau kommt im Organismus, wenn auch in geringem Masse, die zelluläre Resorption des Lipiodols in Betracht, die man bei längerer Versuchsdauer in den Lungenschnitten der Kaninchen hier und da beobachten konnte. (Abb. 6).

Die phagozytierenden Zellen vermögen sowohl korpuskuläre Fettzellen wie auch gelöste Fettsubstanzen aufzunehmen. Die Endothelzellen beteiligen sich an diesem Vorgang in hohem Masse, was auch in unseren Lungenschnitten zu beobachten war. Die Fettropfen werden bald in ihrer Form geändert. Fettsäurenadeln treten auf. Die Endothelien und Adventitiazellen und die Alveolarepithelien zeigen nach einiger Zeit bei Sudanfärbung kleine Fettropfen, was wir in geringerem Masse auch in unseren Versuchen finden konnten. KOJO hat diese Vorgänge genauer untersucht und gefunden, dass zu Beginn der Resorption in der Umgebung eines Embolus viele Leukozyten auftreten, ohne jedoch grössere Fettmengen aufzunehmen. Die ausgiebigste Phagozytierung erfolgt erst einige Zeit später, wie schon erwähnt hauptsächlich durch die Alveolarepithelien und Gefäss-Endothelien. Die Leichtigkeit dieser Vorgänge hängt nach seiner Meinung vorwiegend von der chemischen Verwandschaft des Fettes ab, weshalb erklärlich ist, dass beim Lipiodol alle diese Veränderungen in geringerem Masse vorhanden sind und viel langsamer verlaufen.

ZUSAMMENFASSUNG

Bei der Hysterosalpingographie findet man gelegentlich ein Eindringen des verwendeten Kontrastmittels in die Uterus- und Beckenvenen. Von hier aus gelangt das Lipiodol durch den Blutstrom über die Vena iliaca communis in die Vena cava inferior, wodurch es für kurze Zeit als breites Band parallel der Wirbelsäule röntgenologisch sichtbar wird. Später bleibt das Jodoel in den Lungenarterien und Kapillaren stecken, wo es rasch in seine Hauptbestandteile: Öl und Jod gespalten wird. Das letztere wird als Kaliverbindung zum grössten Teil in den ersten sechs bis acht Tagen vorwiegend durch die Nieren ausgeschieden. Pathologisch-anatomisch sind in den Lungen und übrigen Organen der Kaninchen keine Schädigungen nachzuweisen.

Klinisch wird die Embolie meist symptomlos ertragen. Sie kann aber im Einzelfall, wenn grössere Mengen von Lipiodol direkt ins Gefässystem eingespritzt werden, doch zu schweren Komplikationen, ja sogar zum Tode führen, wenn die heute bekannten Vorsichtsmassregeln verletzt werden.

SUMMARY

On hysterosalpingographic inspection it is sometimes found that the contrast fluid employed has entered the veins of the uterus and the pelvis. From there, the lipiodol is carried with the blood stream, via the common iliac vein, to the inferior vena cava, and thus for a short time becomes roentgenologically visible as a broad band parallel

to the spinal column. Later, the iodized oil gets stuck in the arteries and capillaries of the lung, where it rapidly splits up into its two principal components, oil and iodine. Most of the iodine becomes excreted in the course of the first six to eight days, as potassium compound, chiefly through the kidneys. Patho-anatomically, no lesions can be demonstrated either in the lung or in the other organs of the young rabbits, nor does the embolism, as a rule, produce any clinical symptoms. Occasionally, however, when considerable quantities of lipiodol are injected directly into the vascular system, it may give rise to severe complications, or even be fatal, unless the now well known precautions are duly observed.

RÉSUMÉ

Dans l'hystéro-salpingographie on voit de temps à autres la matière opaque entrer dans les veines de la matrice et du bassin. De là le lipoiodol atteint, en suivant le flot sanguin, la veine cave inférieure après avoir suivi l'iliaque commune. On peut ici le voir un petit instant comme une large bande parallèle au rachis sur l'écran. Plus tard le lipoiodol se disperse dans les artères et capillaires pulmonaires où il se divise vite en ses deux composants principaux; l'huile et l'iode. Ce dernier est en majeure partie évacué par le rein durant les 6 à 8 jours qui suivent, sous la forme d'un composé de potassium. A l'examen anatomique des poumons et autres organes des lapins il n'y a rien de pathologique à retrouver.

Dans la clinique l'embolie est généralement exempte de tous symptômes. Cependant dans quelques rares cas, où une grosse quantité de lipiodol a été injecté directement dans le système veineux, il peut y avoir des accidents graves, même mortels, quand les précautions, connues à ce jour ont été négligées.

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RADIOLOGICAL TREATMENT OF CARCINOMA OF THE ESOPHAGUS[1]

by

Rolf Bull Engelstad

The results obtained in the treatment of carcinoma of the esophagus have so far been very poor, as a permanent cure only rarely has been observed. The histological structure of these tumours is on the other hand of such a kind that a priori we may assume that they are radiosensitive. Generally we have to deal with squamous-cell carcinomas of a higher or lower degree of differentiation, the tumour being occasionally so little differentiated, that it is only with the greatest difficulty one can prove that it is of squamous-cell origin.

In trying to judge the results so far obtained, it is only natural that we first of all consider the primary effect of irradiation on the tumour itself, so as to get an impression of its radiosensitivity. The next problem to consider will be the reason why the permanent results are as bad as they are, and through these investigations find out which ways to choose to improve the results.

At the Norwegian Radium Hospital we have in the years 1932—1938 treated altogether 119 cases of carcinoma of the esophagus. Of these 95 were men and 24 women.

Table I

shows the distribution of patients in the different years:

1932	3
1933	12
1934	8
1935	18
1936	27
1937	29
1938	22

Table II

shows the distribution according to age:

30—39 year	1
40—49 »	9
50—59 »	35
60—69 »	52
70—79 »	21
80— »	1

[1] Opening lecture at the congress of The Northern Association of Medical Radiology, Oslo June 1939.

As will be seen, most of the cases occur after 50 years of age. Approximately $^3/_4$ between 50 and 69 years of age. The average age was 62, the youngest patient being 39 and the oldest 80 years of age.

Of possible enthiological factors, we have first of all fixed our attention on the consumation of alcohol, and regarding women, also to the presence of achylia-anemia and the syndroma of Plummer-Vinson. As these conditions, however, only lately have been the object of systematical investigations, we lack for some parts of our material the necessary informations.

For 25 men we have exact informations regarding their consumption of alcohol. Of these 8 had been moderate consumers and 17 very excessive ones. It is probable that concentrated alcohols (brandy and heavy wines) play the greatest rôle as ethiological factor. It is easily understood that the consumation of these alcohols will produce a chronical irritation of the mucous membrane of the esophagus.

Regarding the frequency of achylia-anemia and the syndroma of Plummer-Vinson present in our women patients, we have come to the following results:

Table III

Certain achylia-anemia or the syndroma of Plummer-Vinson	9
Probable » » » » » » » »	3
No » » » » » » » »	10
Informations lacking	2

Consequently, in half of our female material we have proved the presence of certain or probable achylia-anemia or the syndroma of Plummer-Vinson. In spite of the material being so limited this observation suggests that the conditions mentioned acts as predisposing factors in the carcinomas of the esophagus in the same manner as proved with carcinomas of the oral cavity and pharynx (AHLBOM).

In our material, histologic examinations are made in 83 cases, and in 36 cases not. It is difficult in certain cases to obtain a satisfactory biopsy during the esophagoscopy, particularly where the stenosis is narrow and the opening covered with mucous membrane. It happened to us 15 times, with clinically and roentgenologically diagnosticated cases of carcinoma of the esophagus, that we did not obtain any of the tumour itself, but only the surrounding more or less altered mucous membrane.

The histologic diagnoses were:

Table IV

Poorly differentiated carcinoma	9
Squamous-cell carcinoma	46
Spino-cellular carcinoma	10
Adenocarcinoma	3
	68

The three adenocarcinomas were found in the lowest part of the esophagus. Otherwise, as shown in table IV, squamous-cell carcinomas of very varying degree of differentiation were found, often poorly differentiated and some times quite highly so.

The permanent cures from the treatment are, as before mentioned, very poor. As shown in table V only 10 patients out of our material are, when this it written, still living.

Table V

Living	three	months	after	treatment	5
»	four	»	»	»	1
»	five	»	»	»	1
»	six	»	»	»	1
»	eleven	»	»	»	1
»	fourteen	»	»	»	1[1]

In our entire material we find that the duration of life after the treatment has been as shown in table VI:

Table VI

> 3	months	25
3— 6	»	46
6—12	»	26
1— 2	years	12

Table VII shows the duration of life after the appearance of the symptoms.

Table VII

3— 6	months	25
6—12	»	55
1— 2	years	31
< 2	»	8

It is seen that approximately $^2/_3$ of the patients die in the course of the first 6 months after treatment, and about a corresponding number in the course of the first 12 months after the appearance of the symptoms. The period of living is subject to great variations. Duration of life after the treatment has varied between 1 month (or less) and 21 months, the average duration being 5.1 months. Duration of life after the appearance of the symptoms has varied between 3 months and 41 months, the average duration being 10.9 months.

We will now proceed to discuss the primary effect of irradiation. 8 patients have for different reasons, mostly that of poor general condition,

[1] Free from symptoms but has later got carcinoma of the stomach.

been untreatable. In the remaining 111 patients it has often been difficult to a give complete treatment according to our routine method. But all the same, we will look at the total of this material.

Table VIII

Symptomfree	4
Highly improved	21
Improved	38
Not improved	32
Progression	16

As symptomfree we have only put down patients where the roentgen examination and the esophagoscopy showed a complete disappearance of the tumour for a shorter or longer period of time (fig. 1—6). By highly improved is understood that the swallowing difficulties have practically disappeared, so that the patient is able to take solid food and, consequently, there is a decided improvement of the general condition. As improved we have put down patients where the treatment has had a marked effect, but where the improvement partly has been moderate and partly of a very short duration. The total shows 63 patients (or 56.8 %) who have more or less profited by the treatment. In 16 patients the condition has grown worse during the treatment, and concerning a few of these, this is probably due to the treatment.

The aggravation of the condition has partly consisted of a lowered general condition, often due to the roentgenkater, and partly of swallowing difficulties and pains, probably due to edema in the tumour, and not infrequently rise in temperature which mostly is due to the aggravation of an already exsisting infection in the tumour.

Our post mortem material is very limited but shows all the same the radiosensitivity of these tumours. Post mortem examination was performed in 8 cases, but 2 of these were untreated or not completely treated, and are consequently without interest. Of the other 6, no certain trace of the carcinoma was found in 1 case, great effect of the irradiation was found in 2 cases, and an apparent but moderate effect of the irradiation in 3 cases. Regarding the patient who was symptomfree a noma-like condition had developed and bacteriological investigations showed large numbers of spirochetes and fusiform bacterias. This observation suggests the necessity of an adequate diet with sufficient vitamins. The condition is probably a rare one, even where the alimentation was as problematic as here, and mostly it will be difficult to ascertain the diagnosis in vivo. Any other treatment, eventually a prophylactic one with injections of neosalvarsan will consequently seldom come into consideration.

1. 2.

Fig. 1. Carcinoma in the upper third of the esophagus. The stenosis is nearly complete.
Fig. 2. Same case after treatment. Dose 11,000 r.

In our treatment of the carcinomas of the esophagus we have in a few, altogether 4 cases, administered radium alone, otherwise we have administered roentgen alone, or roentgen and radium combined. The roentgen treatment has been given as a protracted and fractional irradiation with daily doses varying between 150 and 300 r. The amount of the total roentgendoses has been subject to considerable alterations, partly we have had the not desired variations which are determined by the general condition of the patient as well as the local condition of the disease, and partly we have had intentional alterations, as lately we have tried to arrive at much larger doses than before. If it is possible to complete the treatment we now give not less than 12,000 r. distributed on four fields, and if roentgen treatment alone is used, we prefer to go even higher in the dosage. Still, our experience so far, is that additional increase in the dosage often is prevented by the local and general reaction of the patient.

We administer the radium treatment in the following manner: A number of radium tubes (most frequently 3—5 à 10 mgr.) determined

3. 4.

Fig. 3. Carcinoma in the middle third of the esophagus.
Fig. 4. Same case after teatment. Dose 8,000 + 27 Dominici-units.

by the length of the tumour, is fitted inside an esophagus sound. The filtering is 1 mm. platinum + the wall of the sound. This sound is placed in the esophagus under roentgen controle, and stays in place generally 5 hours daily for fram 5 to 7 days, in the cases where a combined roentgen-radium treatment is given. In this way a total dose of 30 Dominici-units is obtained, which converted into r should correspond to about 2,500 r. In the few cases where radium treatment has been administered alone, we have used larger doses, about 40 Dominici-units (corresponding to about 3,500 r). The dose administered in the combined treatment was before often somewhat smaller, about 20 Dominici-units (corresponding to about 1,750 r).

By the method we now employ in combined treatment, the dose that reaches the tumour will be quite large. Supposing we are able to complete the treatment according to our routine method, we may with the roentgen treatment attain a depth dose of about 3,000 r. In addition to this com-s

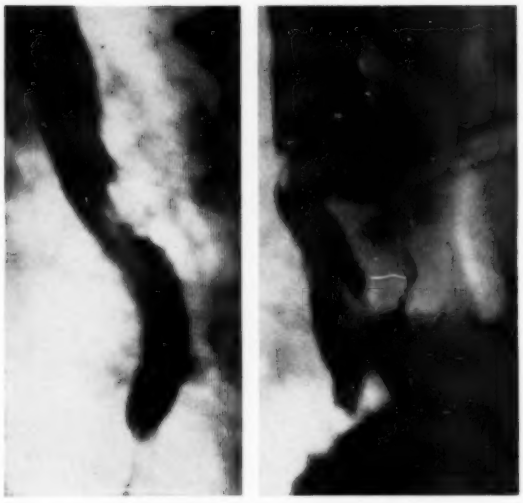
5. 6.

Fig. 5. Carcinoma in the lower third of the esophagus.
Fig. 6. Same caseafter treatment. Dose 10,000 + 30 Dominici-units.

the radium dose corresponding to about 2,500 r. The total dose on the inside of the esophagus should, under favourable conditions reach to approximately 5,500 r. It is, however, evident that the dose reaching the peripheral part of the tumour will be considerably smaller, as in the radium treatment the dose is quickly diminished toward the periphery. The before mentioned dose of 5,500 r represents, therefore, the maximum dose that reaches the tumour.

To complete the combined treatment 8 to 10 weeks are generally required. In many cases the time is longer because shorter or longer periods of suspension is often necessary due to the general and local reaction on the irradiation. Our experience is that we often meet with difficulties before we are able to complete the treatment. In many cases the roentgen dose administered will be too small, and in other cases one is not able to complete the radium treatment, either because the passing of the sound into the esophagus is impossible, due to the stenosis, or because the general condition of the patient is poor.

Before proceeding to study the primary results of the treatment obtained with the different methods and dosages, it will ne necessary to look



7. 8.

Fig. 7. Early case of esophageal carcinoma, polypoid tumour.
Fig. 8. Advanced case of esophageal carcinoma, with large glandular metastases in the neck, but without distinct symptoms from the esophagus.

at the composition of our material, which here denotes the condition of the patient at the beginning of the treatment.

The general condition was only in rare cases such as to be characterized as good.

Table IX

General condition good	24
» » lowered	62
» » poor	33

In $^4/_5$ths of the material the general condition was on admission to the hospital more or less lowered. In more than $^1/_4$th of the cases even so reduced as to be characterized as poor. This condition immediately suggests the difficulties one will have to cope with in completing the treatment.

Gastrostomy has not been performed in more than 23 cases. This auxiliary operation we rarely perform today, even more seldom than before, as generally, it is considered that it does not serve any particular purpose. Only as a rare exception does it facilitate or make the treatment possible, and as a palliative operation it seems to be of small value.

The local findings at the beginning of the treatment has neither been very encouraging in our material. In table X information is given about the spreading of the primary tumours.

Table X

Length	Total	Not circular	Circular
Polypoid	1	1	
< 5 cm	9	5	4
5—10 cm	59	19	40
10—20 cm	48	11	37
> 20 cm	2		2
	119	36	83

The facts that this table shows, regarding the extention of the tumours in length and circumference, are essentially based upon the roentgenological findings. When a tumour is characterized as circular, this does not necessarily signify that it is circular in all its length but only within a shorter or longer part of it. When chiefly based upon the roentgenological findings it can often be connected with some difficulty to decide with certainty, whether the tumour is absolutely circular, or if there is still a little ridge of not infiltrated mucous membrane left, but for the practical estimation it no doubt gives you sufficient knowledge of being confronted with a process, that roentgenologically appears as circular and surely occupies the chief part of the circumference. From this table is also seen, that in our material early cases of carcinoma of the esophagus have been a rarity. The length has been less than 5 cm. in 10 cases only, and one of these came to us at a very early stage. The length was 5 cm. or more in 109 cases, and 10 cm. or more in 50 cases. In 83 cases the infiltration appeared, under roentgenexamination, as circular.

The roentgenological diagnosis of metastases is often connected with great difficulties. We know from experience that the regional lymph nodes frequently are affected, but that they may for a long time stay of a moderate size, which makes it difficult or even impossible to find them at the roentgen examination. In some cases more distant lymphnode metastases may appear at a comparatively early stage, and then first and foremost in the neck. Metastases at this site may even occur in cases of carcinomas that are situated in the lowest part of the esophagus, and may in some cases be the first symptom.

Other metastases i. e. viceral metastases appears comparatively late in the course of the disease. Table XI gives a general view of the frequency of metastases in our material.

Table XI

No metastases proved	59
Doubtful metastases	18
Certain metastases	42

Besides the presence of metastases we must, however, consider another factor which probably plays a great rôle in carcinoma of the esophagus, namely the infiltration of the carcinoma directly into the adjacent tissues. This condition is doubtless of great signification for the attainable results of the treatment, but are rarely accessible for investigation in vivo. Consequently, we have no basis to judge how great a part this condition plays in every individual case.

In classifying the material according to the localization I have, first and foremost, considered it practical to separate the middle third, and will here put down tumours which are found at the height of the bifurcation or immediately adjacent to it. The tumours situated higher up will be counted as the upper third, and the ones situated lower down as the lower third, without having for these last groups described the localization more fully. Of the tumours in the lower third, the ones situated in the cardia, with their chief spreading toward the side of the ventricel, are not counted.

In our material the localization is as seen in table XII.

Table XII

Upper third	31	(26 %)
Middle third	69	(58 %)
Lower third	19	(16 %)

After this general view of the composition of our material we will proceed to search for the conditions which possibly might play a rôle in the results to be achieved. I will overlook here the radiobiological peculiarities of these tumours, and only confine myself to point at what I have previously said of our experiences in this field. The conditions to be considered then will be the following: On one side the manner of growing and spreading of the tumours, their clinical manifestations and their effect on the general condition, on the other side, the possibilities of radiological treatment, the methods to be employed, and the size of the doses to be administered.

Regarding the first group of facts, namely the special characteristics of the disease, we have already mentioned a few important items. We have seen that the general condition of the patient at the beginning of the treatment was more or less affected in $^4/_5$ths of the cases, and that it was extremely poor in more than $^1/_4$th of the cases. We have also seen

that the tumour in most of the cases was very widespread, namely 5 cm or longer in 109 (of 119) cases, 10 cm or more in 50 cases, and circular in 83 cases. Furthermore we have seen that in one half of the cases certain or possible metastases were present.

These conditions must no doubt seem discouraging when we look at the possibilities of the treatment.

The results in the different localizations are seen in table XIII. Patients who are improved are only mentioned here.

Table XIII

	Upper third	Middle third	Lower third
Cases treated	31	69	19
Symptom-free	2	2	
Highly improved	8	8	5
Improved	9	21	8
	19 (61.3 %)	31 (44.9 %)	13 (68.4 %)

It may seem from this table as if the tumours situated in the middle third have been less favourably affected by the treatment, than the ones situated in the upper and lower third, but in working with as limited numbers as here one should be careful and not draw too definite conclusions. For this reason the percentages seen in the table, concerning what can be characterized as favourable results, must not be counted on too much. It is likely, on the other hand, that the tumours situated at the bifurcation can offer some special factors of danger during the radiological treatment, like perforation to the trachea or bronchies, and arrosion of the aorta, factors which either are not present at all, or not to the same degree, in the other localizations. I will mention that perforation to the trachea and bronchies is definitely proved in 5 cases, but the exact number has in all probability been much larger. A few times we have also seen fatal profuse hemorrhages probably due to arrosion of the aorta, but not having had the opportunity of performing autopsies in any of these cases, we have not had our supposition proved. Altogether, our investigations regarding the significance of the localization on the obtainable results, have so far not secured us any valuable facts.

The conditions just mentioned are of such a kind that they are out of our reach and can not be affected by any intervention from our part. The same is the case with the clinical manifestations, that means the manner and the time the disease manifests itself.

For the results of the clinical manifestations, namely the recognition of the disease as early as possible, we might, however, to a certain degree be able to produce a change in a favourable direction. It is seen in cur

material, that at the time when the treatment began, the patient had had noticeable symptoms for a time varying between 1 and 36 months. The average duration of the symptoms had been 5.8 months, or nearly half a year. Through constant educational work we ought to try to improve this condition, but at the same time we must be aware of one important problem that we have to cope with, namely the fact that alarming symptoms apparently do not appear untill the disease is far advanced. Our impression is that the swallowing difficulties do not appear before the tumour is either quite or nearly circular, or before it has reached a size so that the lumen of the esophagus is almost completely filled out. In rare cases the symptoms will appear at an early stage (fig. 7), but as a rule they appear later. Briefly I will state an example of this to show the difficulties connected with the stating of an early diagnosis in carcinoma of the esophagus.

A 71 years old man was admitted to the Norwegian Radium Hospital with the diagnosis tumour colli. At the first examination he denied having had difficulties in swallowing. As a puncture biopsy showed squamous-cell carcinoma, and as no focus could be proved either in the oral cavity, throat or sinuses, he was again submitted to a thourough examination. Finally he admitted having had slight disagreable sensations when swallowing. The symptoms, however, were so slight that he was not even sure of them himself, and when a roentgenological examination of the esophagus was made, he again denied having noticed anything particular when swallowing. Meanwhile, the roentgenological examination of the esophagus showed a carcinoma situated in the lowest section of the esophagus, with a length of 7 cm. (fig. 8). The roentgenological examination also showed a slight restriction of the passage at this site. This case gives an illustrating example of how far a carcinoma of the esophagus may develop before giving subjective troubles.

There is no doubt that increased education on the subject may help in the recognition of the disease at an earlier stage than has been the case, even though the difficulties in stating an early diagnosis can be as hard as in the case just referred to. I therefore consider it very important, with tumours of this type, and in general with malignant tumours, that necessary stress should be put on educational work. In this way, the time that elapses before the patient recognizes the first symptoms and till he comes for treatment, may be considerably reduced. This does not essentially mean that we will be able to state the diagnosis early enough and begin the treatment at an early stage in the development of the process of the disease, because our knowledge of the rapidity in the development at the early stages is too limited. But the possibilities of the treatment will certainly be somewhat more favourable, if the time from the recognition of the symptoms to the beginning of the treatment is to a certain

degree shortened. Under any circumstances it ought to be a necessary nature in our efforts to work for this.

But even if an improvement in the results are achieved in this way, we must consider it our chief field in the future to work for the development and adaption of our radiotherapeutic methods.

I have already spoken of the methods employed at the Norwegian Radium Hospital and have emphazised the considerable variation regarding the size of the doses. These variations have been partly intentional and partly imposed by the condition of the patient.

In quite a number of cases it has been difficult, or even impossible to complete the treatment. The condition was in 8 cases so poor that no treatment at all could be given. In 33 cases the treatment was started but had to be discontinued before we had reached doses that possibly could have had any greater influence on the tumour. In other cases as well, we have been obliged to discontinue the treatment before it was completed according to our routine method, but where we all the same have reached doses that have had some therapeutical effect.

In the following tables I have included all the patients that have got radiological treatment, without considering the size of the doses we have administred or have been able to administer.

Radium treatment alone, with doses of about 40 Dominici-units or more have been administered in 4 cases. This should correspond to about 3,500 r—4,000 r. The results are seen in table XIV.

Table XIV

Total	4
Primarily sympt.free	1
Highly improved	2
Improved	1

In speaking of our post mortem material I have already mentioned the patient who was symptomfree. A noma-like condition which proved fatal developed, but at the autopsy no traces of his carcinoma in the esophagus were found.

We have for the chief part of our material administered roentgen treatment exclusively, namely in 76 cases. Table XV gives the size of the doses and the effect obtained:

Table XV

Dose	Total	Sympt.free	Highly impr.	Impr.	No impr.	Progression.
Incompletely treated (doses less than 5,000 r)	31			5	19	7
5,000—8,000 r	29		2	9	10	8
8,000—12,000 r	14		4	8	1	1
> 12,000 r	2		1	1		

It appears from this table that in some cases even quite small doses may give a subjective improvement, which also has a roentgenologically demonstrable basis. This improvement need not be due to an excessive radiosensitivity of the tumour, but might be due to the fact that the roentgen treatment produces an improvement of complicating infectious processes, and accordingly not a reduction of the tumour itself. We are, however, debarred from getting information about how great a significance is to be put on either of these factors.

As far as can be concluded from the small number, the table also shows us that the primary results are the best in the groups that have received the heaviest doses. It is evident from our material, that if roentgen treatment alone is administered one should try to reach doses of more than 12,000 r and possibly between 12,000—and 16,000 r. From our experiences so far we know that it is difficult to go higher than this on account of the general and local reaction.

Concerning the necessity of very large doses, we have also arrived at the same experiences regarding the group of patients where combined roentgen-radium treatment has been administered. Earlier, this treatment has in most cases been administered in the following manner: We started with the roentgen treatment, and after its termination we placed the radium sound in the esophagus. Of late, however, we have more and more tried to start with the radium treatment for reasons which I later will return to.

In table XVI I have gathered the patients that have received this combined treatment. I have for practical reasons only been able to divide this part of the material into three groups after the size of the doses. Namely I. the incompletely treated, II. patients that have received moderate doses, calculated to a total of between 5,000 and 10,000 r. and III. patients that have received larger doses, a total more than 10,000 r and often even more than 12,000 r. Lately we have made an effort to give a roentgendose of 12,000 r and in addition a radiumdose of 30 Dominici-units, the total of which should correspond to a dose of between 14,000 and 15,000 r.

Table XVI

Dose	Total	Sympt.free	Highly impr.	Impr.	No improvement
Incompletely treated . .	2			1	1
5,000—10,000 r	22	1	9	11	1
> 10,000 r	7	2	3	2	

As previously mentioned, the numbers in our material are so small that one is not justified in making comparisons that may be given statistical value, but we have a decided impression that for the whole of our

material the best primary results are obtained with this combined roentgenradium treatment with heavy doses.

There is another condition which it is necessary to point out, namely, that wether roentgen treatment alone or the combined roentgenradium treatment have been given, we have seen that in not few cases the doses aimed at are not obtained, because the local, and espcially the general condition may prevent this. This again depends on the local and general condition at the beginning of the treatment. We can, for this reason not say that, if we had administered much heavier doses in all cases, the result would have been much better. It is necessary to keep in mind that in a number of cases it is impossible to complete the treatment with heavy doses, and this must be noted on the debit-side of the radiological treatment. It is important to remember this, because our efforts in improving the results must, amongst other things, aim at rendering possible the completion of the treatment with heavy doses in as many cases as possible.

To succeed in this, we must try with all our means to keep the state of alimentation as good as possible, eventual kater must be treated, as well as inflammation that is often present in the tumour. It would also be desirable to reduce the local reaction from the irradiation, but this is hardly possible to any special degree. These points I have to content myself in mentioning only, without going deeper into the subject. I will return to some of these conditions in my conclusions.

In the preceding we have only paid attention to the primary effect of the treatment itself, and have concluded that subjective and objective improvement have occurred in a number of cases. We can, consequently, conclude that the patients have profited by the treatment. But we should not content ourselves with the knowledge that the troubles of the patients have reduced, but try to find out what influence the treatment has had on the longevity, counting from the appearance of symptoms. It has not been easy to find a material suitable for this investigation as patients who have never received radiological or surgical treatment can only be considered, and besides this we must have exact informations regarding the progress of the disease.

From the post mortem records of the pathological laboratories of »Rikshospitalet», Oslo, I have in the years 1926—1937 found 25 cases that satisfied these demands. The period of living for these patients have on the average been 6 months after the first appearance of the symptoms, the limits being 3 and 12 months. The corresponding average-number in our material is 10.9 months, the limits being 3 and 41 months. Though reservations again have to be made for the small number, we can take into account that, concerning the longevity, the patients have pro-

fited by the radiological treatment, and they certainly have, at least in many cases, had a life more worth living.

In view of the present conditions, concerning the treatment of carcinoma of the esophagus, our chief interest must necessarily be to concentrate ourselves on the possibilities that a continued work with this disease will involve. And the question arises, what informations can our present material give us in this respect.

Our present knowledge of carcinoma of the esophagus, does not make it probable, that we will be able to reduce essentially the frequency of the disease with prophylactic measures. We might perhaps prevent some cases of carcinoma of the esophagus, by treating achylia-anemia and the syndroma of Plummer-Vinson as early and energetically as possible, and something might perhaps be achieved by discouraging the abuse of alcohol and warn against the use of too hot, too cold or too spicy foods, but our achievements in this direction will undoubtedly be very limited. But new possibilities in these fields should continually be looked for. The study of the causal factors should be as important to us clinical workers as to the experimentally working investigators. We have, however, no scientific basis for the establishing of a more extensive prophylaxis, and are for this reason obliged in our practical work to concentrate us first and foremost on an effective utilization of our diagnostic and therapeutic methods.

It is seen that most of the patients come for treatment with a far advanced local process. Half or more have in addition metastases and most of them have a reduced or often poor general condition. Being at the same time aware of that the patients do not come for treatment before the symptoms have been present for several months, on the average 5.8 months, it is evident that one of our chief tasks should be an effort to make an earlier diagnosis possible, both by educational work amongst the public and by improving and utilizing, as far as possible, our means for stating an early diagnosis. Even so, it will not be easy to start the treatment early enough, but it may in spite of all mean a small improvement in the right direction. It is, however, to be feared that the improvements achieved by these means will be of a comparatively moderate extension.

Our chief problem to-day will be how to treat the fully developed and often far advanced disease, most efficiently. I have not mentioned before the possibilities of a surgical therapy, and will not discuss the problem here. It is sufficient to ascertain the fact that so far results are not obtained that can bring this method in the foreground. First of all the risk run is too great and the possibilities of a radical operation too small.

Therefore, our chief task is to increase the possibilities for radiologic treatment and develop our radiotherapeutic methods to their greatest

capacity. Our material shows that it is necessary to give very heavy doses, but it also shows, that the completion of the treatment with this method is not always possible. The obstacles that prevent the completion of the treatment are partly due to the disease as such, or to the general and local reaction on the irradiation, and partly to the complications that are either present in advance or appear during the treatment.

The local reaction on the irradiation can of course not be avoided, and the question will only be, how it may be possible to reduce its importance as an obstacle to the treatment. The complications from the local reaction are partly pains and swallowing difficulties, and partly a restriction in the passage due to hyperemia and edema. I have mentioned that the treatment requires 8 to 10 weeks or more when completed according to our routine method. Possibly, it would prove an advantage if we could shorten this time, in other words, give a more concentrated treatment, in order to utilize to its full extent the time of latency. We have not tried this method, and I have for this reason no decided opinion of its value, but we may assume that the most annoying local reaction will be transferred towards the termination of the treatment or even after it. How much one will be able to shorten the treatment is on the other hand quite limited.

Apart from the conditions mentioned there is another consequence due to the direct effect of irradiation, namely the local reaction in the trachea, bronchies and lung. This reaction may sometimes lead to an often quite troublesome cough, which however as a rule can be checked by current cough remedies. In the lungs we do not often see a pronounced, roentgenologically demonstrable reaction on the irradiation, probably because our fields of radiation are placed as close to the midline as possible. Bronchopneumonias that are often seen, especially in the advanced stages are hardly due to the irradiation, but in all probability mostly regular aspiration pneumonias.

As to the general reaction it is the pronounced forms of kater, as well as the changes in the blood, first and foremost the leukopenia, that are of importance. In the treatment of kater, Ephetonin preparations as well as B-vitamin and liver preparation have been administered. The greatest benefits has been derived from the liver preparations, in some cases it has been quite astounding. In the treatment of carcinoma of the esophagus, the leukopenia has seldom been an obstacle for the completion of the treatment. When it does happen, an interval in the treatment is necessary. We have in these cases, as well, administered very large doses of liver preparations, but it is hard to say if they have had any noticeable effect. A few times, in the case of leukopenia which have appeared during treatment in other forms of carcinoma, we have administered Nuclosin, but we seldom administer this remedy any more, as we have seen very

serious shock-like reactions after the injection, even when this has been given carefully and in refracted doses.

The complications that arise are often very difficult to treat. Primarily this concerns perforation to the neighbouring organs and the occasional hemorrhages. In these cases one is as a rule quite powerless. But also the infection which is often present, is mostly difficult to treat and does often necessitate long intervals in the treatment. In a few cases we have tried some of the chemo-therapeutical remedies, like Prontosil and Streptan, but without convincing results. On account of our before mentioned experiences about fuso-spirochaetal infection we have a few times tried to administer neosalvarsan in careful doses, but this too has been without any certain effect. The most important item in the treatment of the infection will, therefore, be to postpone the treatment and at the same time carry out a lenient regimen.

Of greatest importance to us are the obstacles for the completion of the treatment which are produced through the process of the disease, namely restriction in the passage and consequently reduced alimentation possibilities, and cachexia. The restriction in the passage will often be quite pronounced at the time when the patient comes for examination, but otherwise it varies between being hardly a noticeable inconvenience and a complete stenosis. In the more pronounced degrees of stenosis the performing of a gastrostomy may seem tempting and in some cases vitally indicated. Lately, however, we have performed this operation very seldom as we are under the impression that its value is very limited. In the cases where it has been performed the state of alimentation has all the same been difficult to keep up.

In most cases we content ourselves with giving the nourishment as abundant and adequate as possible. Liquid or soft food, supplemented with nutritive enemas, if necessary, must be given. The composition of this nourishment I will not discuss any further, but only mention that our efforts have been to meet the usual demands of a hygienic correct alimentation, and have considered a sufficient supply of vitamins as indispensable.

In some cases it may happen that the stenosis gets worse in the course of the treatment. This is mostly due to the fact that the tumour does not react to irradiation, or that it gets time to progress before effective doses have been reached. This again, may in the course of the treatment lead to conditions of emaciation and cachexia, and will make a suspension of the treatment necessary. In our efforts to assure as good a passage as possible during treatment, we have lately, in cases where this has been possible, started with the application of the radium sound and proceeded with roentgen irradiation. Our intention with this method is to get a destruction of the tumour masses, which are prominent in the

lumen, and of the most superficial layer of the tumour infiltration, in order to reduce the stenosis. We have not sufficient experiences to enable us to judge the value of this method, but it seems quite rational to us.

Considering all experiences gained, we now prefer to complete the radiological treatment as follows: If possible, we first apply the radium sound and give in the course of from 5 to 7 days a dose of about 30 Dominici-units, corresponding to about 2,500 r. We then give roentgen treatment, with daily doses of between 150 and 300 r, mostly 250—300 r., with total doses of about 12,000 r or more, even 16,000 r is given where this is feasible. In the future we may even try to increase the daily doses still further, in order to shorten the duration of the treatment. The roentgen irradiation is given with 175 kv. and 4 ma. filter 2 mm. Cu., focus-skindistance 50 cm. 4 fields are irradiated, 2 in front and 2 in the back, both close to the midline. This last to avoid as far as possible the irradiation of the lungs and also to facilitate the adjustment. The length of the fields is dictated by the tumours extension in the length, the width is generally not more than 6—10 cm. It may be connected with great difficulties roentgenologically to prove the length of the process of disease. We had a striking example of this at an autopsy performed a short while ago. In a distance of 4 cm. from the roentgenologically proved and now visible and palpable boundary, we could ascertain a decided epithelial hyperplasia with signs of malignancy. Therefore, the fields should be made considerably larger than indicated by the length which is proved roentgenologically.

It is evident from what I have stated here, that our material does not permit us to indicate that one of the methods employed in the radiological treatment is superior to the other, but it seems as if the primary results are better in the combined roentgen-radium treatment. That very heavy doses are necessary we can conclude with certainty. In order to be able to accomplish this, the alimentation has to be closely watched, and the general and local reaction as well as eventual complications, must be treated to the best of our ability. This auxiliary treatment, if it may be termed so, is a necessary foundation in the treatment of carcinoma of the esophagus and will surely be of great value for the results to be obtained.

SUMMARY

119 cases of esophageal carcinoma have been treated at The Norwegian Radium Hospital during the years 1932—1938. Permanent cure has not been obtained in any case, but the roentgen examinations as well as the few post mortem examinations have proved that the tumours most often are, to a smaller or greater degree, radiosensitive. Of the whole material 111 patients have been treated, but the treatment has in 33 cases been quite incomplete. The primary results are: Symptom-free 4, highly improved 21,

improved 38, not improved 32, progrediating 16. The average duration of life was 5. months after the treatment and 10.9 months after the onset of symptoms.

If possible a combined radium-roentgen treatment is given. Our present routin method is: the radium is placed in a sound in the esophagus and we give a total dose of 3 Dominici-units (corresponding to about 2,500 r). The roentgen treatment is given from fields, with daily doses from 150 to 300 r and a total dose of 12,000 r or more. In the case treated only with roentgen, a total dose of 12,000—16,000 r is desired. With the combine treatment the maximum depth dose obtained on the inside of the esophagus may be up t 5,500 r. To complete the treatment 8—10 weeks are required.

Most of the cases come for treatment with a far advanced disease, only one earl case has been seen. Very often it is impossible to give as large doses as desired.

The combined radium-roentgen treatment with heavy doses seems to give bette primary results than roentgen treatment alone. The necessity of very heavy doses may b regarded as certain.

In order to be able to accomplish this, the alimentation has to be closely watched, and the general and local reaction as well as eventual complications must be carefully treated.

In the paper is given a detailed analysis of the material, of the different methods o treatment and their results and of the possible ways to go in order to improve the results.

ZUSAMMENFASSUNG

In den Jahren 1932—1938 wurden am Norwegischen Radiumspital 119 Fälle von Ösophaguskarzinom behandelt. In keinem Fall ist Dauerheilung erreicht worden, aber die Röntgenuntersuchungen wie auch die wenigen post mortem-Untersuchungen haben erwiesen, dass die Tumoren meistens in geringerem oder grösserem Grade radiosensitiv sind. Vom Gesamtmaterial wurden 111 Patienten behandelt, in 33 Fällen war die Behandlung aber ganz unvollständig. Die Primärresultate waren: Symptomfrei 4, sehr gebessert 21, gebessert 38, nicht gebessert 32, im Fortschreiten begriffene 16. Die durchschnittliche Lebensdauer betrug 5.1 Monate nach der Behandlung und 10.9 Monate nach dem Einsetzen der Symptome.

Wo es möglich war, wurde kombinierte Radium-Röntgenbehandlung gegeben. Unsere jetzt gebräuchliche Methode wird folgendermassen ausgeführt: Das Radium wird in einer Sonde in den Ösophagus gebracht, und wir geben seine Gesamtdosis von 30 Dominici-Einheiten (ungefähr 2,500 r entsprechend). Die Röntgenbehandlung wird von 4 Feldern gegeben, mit täglichen Dosen von 150 bis 300 r und bedarf es einer Gesamtdosis von 12,000 r oder mehr. In den Fällen, die nur Röntgenbehandlung erhalten, bedarf es einer Gesamtdosis von 12,000 r bis 16,000 r. Bei kombinierter Behandlung soll die Maximal-Tiefen-Dosis auf der Innenseite des Ösophagus bis 5,500 r betragen. Für die vollständige Behandlung sind 8—10 Wochen notwendig.

Die meisten von den Fällen kamen in weit vorgeschrittenerem Krankheitsstadium zur Behandlung, nur ein früher Fall war unter ihnen. Sehr oft ist es unmöglich, so grosse Dosen zu geben, wie es wünschenswert wäre.

Die kombinierte Radium-Röntgenbehandlung mit massigen Dosen scheint bessere Primärresultate zu geben als Röntgenbehandlung allein. Die Notwendigkeit sehr massiger Dosen ist als erwiesen zu betrachten.

Um dies durchführen zu können, ist die Ernährung genau zu überwachen, und sowohl allgemeine wie lokale Reaktion und auch eventuelle Komplikationen müssen sorgfältig behandelt werden.

Verf. gibt in seiner Arbeit eine ins Einzelne gehende Analyse des Materials, der verschiedenen Behandlungsmethoden und ihrer Resultate und der möglichen Wege zur Verbesserung der Resultate.

RÉSUMÉ

119 cas de cancer oesophagien ont été traité au Radium-Hôpital Norvégien durant les années 1932—1938. Une guérison permanente n'a jamais été obtenue, mais les examens radiologiques, ainsi que les rares autopsies, ont prouvé que les tumeurs sont dans la plupart des cas, plus ou moins radiosensibles. Sur tout le matériel 111 sujets ont été traités, mais le traitement a été incomplet dans 33 cas. Les résultats primaires sont: exempts de symptômes 4, considérablement améliorés 21, améliorés 38, non améliorés 32, progressifs 16. La durée moyenne de la survivance fut 5.1 mois après le traitement, et 10.9 mois après l'apparition des symptômes.

Si possible on donne un traitement combiné de radium et de roentgen. Notre méthode routinière actuelle est: Le radium est placé dans une sonde dans l'oesophage et nous donnons une dose globale de 30 unités de Dominici (soit environ 2 500 r). Le traitement par le roentgen est donné dans 4 champs, avec une dose quotidienne de 150 à 300 r, et une dose globale de 12 000 à 16 000 r est désirable. Par le traitement combiné la dose profonde maximum obtenue à l'intérieur de l'oesophage doit être de près de 5 500 r. 8 à 10 semaines sont nécessaires pour effectuer le traitement. La plupart des cas viennent au traitement dans un stade fort avancé, un seul cas récent a été vu. Très souvent il est impossible de donner une aussi grande dose qu'on le désirerait.

Le traitement combiné de radium et de roentgen à fortes doses semble donner de meilleurs résultats que le traitement au roentgen seul. La nécessité de très fortes doses doit être considérée comme certaine.

Pour pouvoir l'accomplir l'alimentation doit être minutieusement reglée, et les réactions locales et générales, ainsi que les accidents éventuels, doivent être soigneusement traités.

Dans l'article on retrouve une analyse détaillée du matériel, des diverses méthodes de traitement et de leurs résultats, ainsi que des voies à suivre pour améliorer ces résultats.

FROM THE ROENTGEN DIAGNOSTIC DEPARTMENT OF LUND HOSPITAL, SWEDEN.
CHIEF: HANS HELLMER, M. D.

THE ROENTGEN DIAGNOSTICS OF PERISINUOUS ABSCESSES[1]

by

Sölve Welin

Ever since the introduction of oto-radiology the sinus sigmoideus has attracted a great deal of interest. Thus in 1907 it was pointed out, for example, by WINCKLER how important it was for the operator to know the position of the sinus already before the operation, particularly when located laterally or anteriorly.

Having been a purely anatomico-topographical question in the beginning the pathological questions are now in the centre of interest.

The first scholar who dealt with the roentgen diagnosis of perisinuous abscesses was LAW, the American radiographer. In a paper read in 1916 this author pointed out that normally the anterior wall of the sinus appears in the radiograph although no traces of the sinus can be seen when the mastoid process is completely destroyed and the sinus exposed.

When publishing his paper the following year he illustrated it by a radiograph of a perisinuous abscess located close to the upper bend of the sinus.

In his comprehensive monograph, »Mastoids» published 3 years later (1920), he had still further improved his diagnostics. Now he distinguished between abscesses located superficially, cortically, and those lying deep down.

In the former case the anterior wall of the sinus, which, as already pointed out by WINCKLER, does not exactly coincide with the posterior border of the pyramid, is said to appear in the radiograph while in the latter case, with the perisinuous abscess located deep down, it would be entirely missing. These signs, however, would only apply in cases where the wall of the sinus is either definitely thick or very thin.

Strangely enough, however, it seems as if LAW's publications had escaped all attention by subsequent workers on this subject.

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In 1923 it was stated by ALBERT HEINEMANN that he had sometimes been able to show defects of the posterior border of the pyramid in cases of perisinuous abscesses. In 8 cases out of 12 such a defect had been discovered while in the remaining 4 cases none could be observed. The author however omitted to offer any explanation of this latter phenomenon.

The same view with regard to the radiographic signs of perisinuous abscess was also held by ERSNER who in 1924 published an article based upon 142 operated cases of acute mastoiditis. A similar opinion was also maintained by TAYLOR who recommended a careful examination of the sinus wall as a defect here pointed to the presence of a perisinuous abscess.

In 1930 BOENTE published an article on the roentgen diagnosis of these abscesses, basing it on 9 cases verified by operation. He distinguished between 2 different kinds of roentgen findings.

Firstly those cases where at the abscess over the sinus the posterior pyramidal border appeared more distinctly than in surrounding regions — thus a view quite contrary to those earlier mentioned.

Secondly such more advanced cases, where the destruction could be seen along the whole of the sinus with a small rarified part over the sinus wall itself; this then would correspond to the 'breach'.

In his academic treatise published in 1936 BRÖSTE paid a great deal of attention to the roentgen diagnostics of these abscesses. After having shown experimentally that the posterior pyramidal border and the anterior wall of the sinus do not coincide — a phenomenon that as a rule is of less importance in practice although already emphasized by Voss and, inter alia, beautifully illustrated in MAYER's monograph — BRÖSTE pointed out that the most important criterion of a perisinuous abscess was the fact that the posterior pyramidal border over a small and limited area appeared more distinctly than in the surroundings; this he considered due to the destruction located in front of and behind the posterior border of the pyramid.

On the other hand he did not agree with BOENTE that the diagnosis can be made with the abscess located behind the posterior pyramidal border, this because the bone here is not homogeneous; wherefore, even normally, areas of greater or lesser density are obtained.

A number of authors however have come to the conclusion that it is impossible to diagnose a perisinuous abscess by roentgen. Thus for example it was stated by BERNHARD HEINE, 1925, in RIEDER-ROSENTHAL's textbook that deeply situated abscesses along the sinus could not be made to appear in the radiograph since they were often covered by compact bone.

In 1933 BERG and CONSTANS gave an account of »clinical, roentgenological and operative findings» in 158 cases of mastoiditis and maintained they were unable to detect any changes near the sinus which might indicate an abscess.

It was further pointed out by W. LOEPP in 1936 how valuable it would be, could one by radiography before onset of clinical symptoms discover the presence of a perisinuous abscess. At the same time he pointed out the great difficulties in making a diagnosis since the abscess sometimes may be hidden from view without ever really reaching the sinus. On the other hand a destruction bordering on the sinus wall would not appear in the radiograph, if covered by compact bone and if the sinus penetrated deeply into the pyramid.

We are not told however if and how often he was able to make this diagnosis and, if so, on what roentgenological changes the diagnosis was based.

In the same year LEONOR BENSCH gave an account of his experiences from the Leipziger Clinic during 1933 and 1934 and maintained that a perisinuous abscess could but exceptionally be diagnosed in the radiograph since it caused a far too faint contrast shadow.

It is then evident that the views held as to the possibility of diagnosing these abscesses by radiography are greatly at variance; and if we disregard those authors that are unable to make this diagnosis, we shall find some who claim a *defect* of the posterior pyramidal border while others refused to make a diagnosis unless the posterior pyramidal border appears more distinctly and better defined at the seat of the abscess than elsewhere.

Diametrically opposed demands have thus been put forth by different authors and all have sustained their views by cases verified by surgical operations.

The question must then be raised whether it would not be possible in some way or other to co-ordinate these views. With a starting-point from the Lund material this is really found to be the case.

At the Lund Hospital in the course of the past 5½ years we have had 44 cases of acute mastoiditis where at the operation performed at the latest 3 days after the radiographic examination one could find one, or occasionally, several perisinuous abscesses. In but 15 cases however was roentgenological diagnosis made prior to the operation since my attention had not earlier been directed to this detail. In all the cases symptoms and clinical observations were of a nature to make the diagnosis of mastoiditis relatively certain. The majority of patients, on the other hand, had not any direct signs of sinus affection, wherefore the roentgenological diagnosis of perisinuous abscess in most cases came as a surprise.

In carrying out an after-examination of the films I found that the perisinuous abscess appeared in 39 cases in radiographs taken in latera

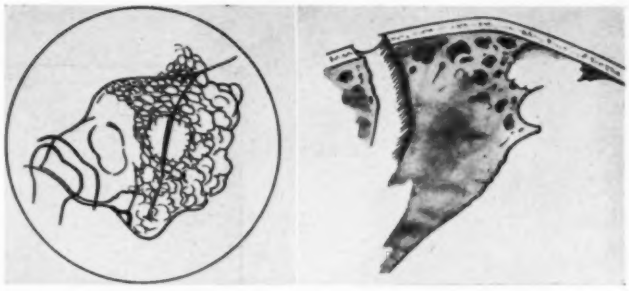

Fig. 1. Diagram of an antero-superficial perisinuous abscess. On the left-hand side: the cell system in RUNSTRÖM's first projection with destruction over middle part of sinus. The posterior pyramidal border appears in this area far more distinctly than elsewhere. On right-hand side: a cut section in the horisontal plane level with the destruction and the sinus partly laid bare.

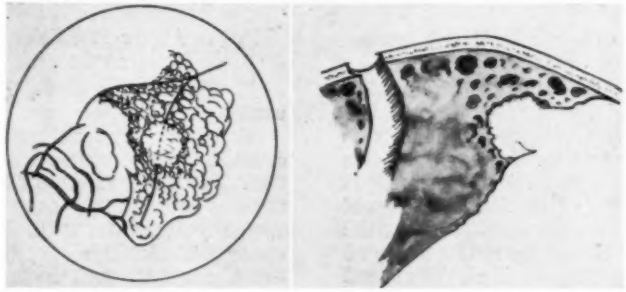

Fig. 2. Diagram of an antero-deep perisinuous abscess. The posterior pyramidal border appears little or not at all level with the abscess. — The section to the right shows the abscess deep down with a defective sinus wall and the pyramidal border decalcified.

projection or in the axial orthoprojection in lateral position, while in the remaining 5 cases no such abscess could be found. I wish here to stress the fact that in these five cases the defect was located on the bulb and that I in examining these five cases have not hitherto been able to discover it on the radiograph.

As to the 39 clear cases a common feature of them all is that the radiograph reveals a destruction of the cellular system in immediate connection with the sinus wall and that stereoscopically this wall presents such a limited and rarified area as to necessitate the presence of a true defect. The great difference in contrast between the rarified area and the surrounding bone excludes the possibility of these changes being explained by any inhomogeneity of the bone.

Fig. 3. Antero-superficial perisinuous abscess.

R. F. Female, aged 18. One month's history.

Roentgen. Small air-containing cells of normal extension. Diffuse infiltration over cellular system. Over the middle part of the sinus there is a large area of destruction extending forwards toward the antrum. In the centre of the destroyed area there is a fairly localised, well marked destruction of the sinus wall, the size of the tip of the finger. Within the anterior portion of this destruction the posterior border of the pyramid is projected, appearing here more clearly and better defined than elsewhere.

Roentgen diagnosis: extensive destruction with antero-superficial perisinuous abscess.

At operation two days later a cavity was found, the size of a hazelnut, filled with pus and at the site indicated by the roentgen examination; sinus exposed over an area the size of the tip of the little finger.

Fig. 4. Antero-superficial perisinuous abscess.

K. O. Male, aged 7. History 3½ weeks.

Roentgen. Pneumatisation of small cells of normal extension. Diffuse infiltration. Not far from the sinus-dura angle there is seen over the sinus a destroyed area, the size of the tip of the finger, in the centre of which appears a destruction of the sinus wall, the size of a brown bean. The posterior pyramidal border is projected through the rarified area and appears here more distinctly than in the surroundings.

Roentgen diagnosis: antero-superficial perisinuous abscess.

At the operation immediately carried out there was complete verification of the roentgen findings.

The above mentioned changes are decidedly the most important one when it is a question of diagnosing a perisinuous abscess, while the changes along the posterior pyramidal border are only a secondary phenomenon which in itself does not permit the making of such

Fig. 5. Antero-deep perisinuous abscess.

S. L. Female, aged 27. History 2½ months.

Roentgen. Pneumatisation exceedingly plentiful. Diffuse infiltration. Multiple destructions along the whole sinus with at least 2 rarefactions not far from the sinus-dura angle, so well marked and localised, that a destruction of the sinus wall must be reckoned with. The posterior border of the pyramid is projected through the uppermost of these rarefactions and is nearly entirely absent.

Roentgen diagnosis: antero-deep perisinuous abscess.

Operation the following day: liquefied areas in accordance with roentgen examination, sinus exposed but covered by granulations.

Fig. 6. Antero-deep perisinuous abscess.

B. G. Male, aged 20. History 3 weeks.

Roentgen. Pneumatisation of normal extent. Diffuse infiltration. Destroyed area over sinus level with the external auditory canal and extending up near the sinus-dura angle. In the centre of the destroyed area a localised destruction of the sinus wall. Posterior pyramidal border here is deficient.

Roentgen diagnosis: antero-deep perisinuous abscess.

Immediate operation verified the roentgen findings.

diagnosis. These changes are obviously only present in those cases where the abscess is located so far forward that the posterior border of the pyramid is projected through the destruction.

When the perisinuous abscess within the anterior aspect of the sinus extends in front of the posterior pyramidal border and is situated superficially under the corticalis, then the pyramidal border appears more distinctly level with the abscess than elsewhere since the rays in the abscess region with its decalcified and deficient cellbars have to penetrate a less amount of absorbent tissue than in the surroundings. This causes the posterior pyramidal border to appear more clearly at the seat

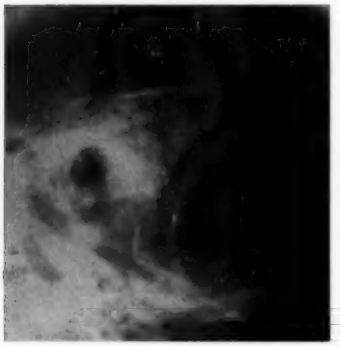

Fig. 7. Posterior perisinuous abscess.

H. P. Female, aged 24. History 4 weeks.

Roentgen. Pneumatisation of small cells of normal extent. Diffuse infiltration. Large destroyed area over posterior part of sinus with destruction of the sinus wall, the size of a hazelnut.

Roentgen diagnosis: posterior perisinuous abscess.

At operation 2 days later roentgen findings were verified.

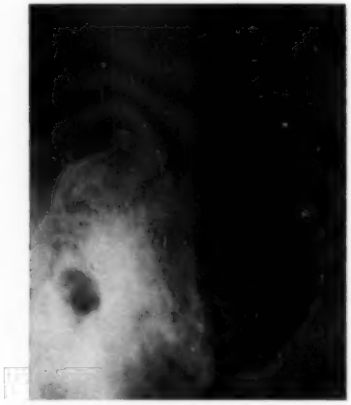

Fig. 8. Posterior perisinuous abscess.

E. N. Male, aged 17. History a fortnight.

Roentgen. Plentiful irregular pneumatisation with large marginal cells over the sinus. Diffuse infiltration. Large destroyed area over posterior part of sinus with a somewhat irregular destruction of the sinus wall, the size of about a hazelnut, level with the antrum.

Roentgen diagnosis: posterior perisinuous abscess.

Operation the following day showed complete agreement between roentgen and operative findings.

of the abscess than elsewhere (vide subjoined table). BOENTE's and BRÖSTE's views also thereby find their explanations.

If, on the other hand, the destruction should extend more deeply, causing the pyramidal border to come more or less intimately in contact with the pathological process, then it will at once appear in a less satisfactory manner. This would then depend upon decalcification or possibly sometimes upon true liquefaction of the pyramidal border. It is true that thereby the roentgen rays will be absorbed to a lesser extent at the seat of the abscess, but as at the same time the pyramidal border is engaged in the pathological process it will nevertheless appear less prominently than in the surroundings. The views held by HEINEMANN, ERSNER and TAYLOR would thus find their explanation. (Vide p. 49)

It should however once more be emphasized that the changes in the posterior pyramidal border are a secondary phenomenon and that the

most important criterion is the direct observation of the defect itself in the sinus wall. With the aid of the pyramidal border, however, we are able more easily to distinguish between three different fully typical pictures of the perisinuous abscesses entirely determined by their different localisations: posterior, antero — superficial and antero — deep.

The diagnostics of these abscesses is in a very particular degree dependent upon a high picture quality and it is thanks to the considerable roentgeno-technical improvements of recent years that the advancement of this very sphere of roentgen diagnostics has been made possible. The greatest credit here is due to the introduction of roentgen tubes with rotary anodes since by these tubes we not only approach the punctiform representation but are also able considerably to shorten the exposure, both factors which of course greatly contribute to the production of sharply defined pictures.

The use of a focussed secondary diaphragm with adjustable speed is also of great value since thereby we avoid all disturbing striping of the film although the time of exposure is usually kept to about one second or less.

Naturally we also make use of the special skull table devised by LYSHOLM and examine the patients in the horisontal position, this being the best means for securing a good fixation.

Lastly it may be mentioned that besides RUNSTRÖMS' positioning of the tube — the abscesses being clearly in evidence in his first (15° cranial tilting of the tube) as well as in his second position (35° cranial tilting of the tube) — we also make use of the symmetrical survey picture according to ALTSCHUL.

SUMMARY

After a brief historical review the author describes the roentgenological changes revealed in perisinuous abscesses and in so doing he takes the opportunity of co-ordinating the diametrically opposed demands on their radiographic appearances earlier entertained by various authors.

ZUSAMMENFASSUNG

Nach einem kurzen geschichtlichen Überblick beschreibt Verf. die durch Röntgenuntersuchung bei perisinuösen Abszessen ersichtlich gemachten Veränderungen und ergreift dabei die Gelegenheit, die diametral entgegengesetzten Forderungen, die früher von verschiedenen Verfassern an das radiographische Aussehen dieser Veränderungen gestellt wurden, in Einklang zu bringen.

RÉSUMÉ

Après un bref exposé historique l'auteur décrit les altérations radiologiques observées dans les abcès périsinusiques, et en le faisant, il profite de l'occasion pour coordonner les exigences diamétralement opposées des différents auteurs sur leurs aspects radiographiques.

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FROM THE ROENTGEN DEPARTMENT AT THE SERAPHIMER HOSPITAL, STOCKHOLM.
CHIEF (ACTING): ERIK LYSHOLM, M. D.

ARTERIOGRAPHIC DIAGNOSIS OF MALIGNANT GLIOMA[1]

by

H. Hemmingson

Roentgenological diagnostics of cerebral tumours has in recent years attained greater and greater perfection and thanks to an improved instrumentarium and a gradually more refined technique it has gained a high degree of certainty. This applies particularly to the localisation of cerebral tumours which by encephalography or ventriculography can be localised with great confidence in the cerebrum as well as cerebellum, central ganglia and brain stem, 3rd and 4th ventricles. Cerebral angiography with thorotrast, as elaborated by MONIZ and LÖHR, has however in recent years been more and more employed for the localisation of expansive processes in the cerebral hemispheres and seems to yield good results, particularly in the cases of tumours of the frontal and temporal lobes (MONIZ, LÖHR, FISCHER). Even expansive processes of the posterior fossa of the skull one has tried to localise by arteriography of the subclavian artery (MONIZ) or by direct opaque injection of the vertebral artery (SJÖKVIST). The method of this arteriography of the vessels of the cerebellum is however rather difficult to carry out and the pictures obtained are fairly hard to interpret because of the fine bore of the vessels and the consequent slight density obtained in displaced vessels or, for instance, in an aneurysm. Cerebellar angiography has not therefore come very much into use. Nor do the tumours located in the brain stem and the 3rd ventricle produce vascular displacement of a kind that permits exact localisation of any expansive tumours situated here. The usefulness of arteriography in local diagnostics is therefore limited to expansive processes of the cerebral hemispheres. More recent investigations have also shown that cerebral angiography with thorotrast is not a harmless procedure (LINDGREN and EKSTRÖM). The thoro-

[1] Submitted for publication Sept. 20, 1939.

trast, a colloidal solution of thorium dioxide, does not leave the organism but is deposited in the reticulo-endothelial system where it remains for years, perhaps for the rest of life. On account of the radioactive properties of thorium damage to the parenchyma may arise, particularly in liver and spleen, in whose reticulo-endothelial cells a powerful depositing takes place. Moreover LINDGREN and EKSTRÖM have shown that in 21 out of 35 brains examined there was retention of thorotrast in the vessels, partly in the form of thorotrast drops swimming about freely in the blood, partly as intravascular thorotrast rods which in 6 cases by embolic closure of fine arterial branches had given rise to cerebral malacia. In these latter cases LINDGREN and EKSTRÖM maintain that some importance must be ascribed to the arteriography as a contributary cause of death. Their investigations also seem to bear out that the risk of endovasal retention of thorotrast in the brain rises with the amount of opaque substance injected, wherefore it would seem of importance to reduce the amount of thorotrast as far as possible.

As regards the technique of angiography employed by us I shall only mention here that after exposure of the carotid bifurcation the injection of the contrast agent is as a routine made into the internal carotid artery. To obtain arterio- as well as phlebogrammes 6 cc. of thorotrast are quite sufficient. On injections into the common carotid a greater amount of thorotrast is required to obtain the same amount of filling of the branches of the internal carotid. The arteriogram is taken towards the end of the injection, the phlebogram 3 seconds after completion of the injection. Since we think such valuable data are obtained from a phlebogram this is to our mind necessary for a complete examination. If it is a case of diagnosing the nature of, for example, a malignant glioma we are content with a lateral picture only; if angiography is carried out for localising a tumour both a frontal and a lateral picture are required.

In up-to-date roentgen diagnostics of cerebral tumours one must not, however, be content with the mere localisation of the tumour. For a proper assessment of indications as well as prognosis as also for planning an operation, it is of the greatest importance for the surgeon to form an opinion as to the nature of the tumour and the degree of its malignancy; here roentgen examination may in many cases do great service. Even a flat film examination of the skull affords us in many cases information as to the presence of a cerebral tumour as well as its location and nature. The of old wellknown pressure erosions in the sella region, sutural ruptures and increased depressions prove the presence of an intracranial pathological process giving rise to increased pressure. The displacement of a calcified corpus pineale — to be studied in the sagittal as well as the frontal direction — helps us to localise an expansive

process. Calcified tumours are usually found in cases of benign glioma, especially in oligodendroglioma, as also in vascular malformations and inflammatory processes, in every case indicating the position of the pathological process. Sometimes it is even possible to draw certain conclusions from their appearance as to the nature of the calcified process. Thus do we find in Sturge-Weber's disease quite characteristic gyriform calcifications, usually situated occipitally (BERGSTRAND—OLIVECRONA—TÖNNIS); if bilateral, such calcifications may be regarded as pathognomonic of this disease. The oligodendroglioma may present the same gyriform structure of the calcifications if these are located in the cortex but naturally they are then localised to the tumour (LINDGREN). Vascular calcifications in arterial aneurysms and vascular malformations may also through their appearance give an indication of the nature of the calcified process.

But in other cases, too, a flat film examination may afford us valuable information for diagnosing the precise nature of the tumour. Local changes of the vascularisation of the skull-cap in the form of irregularly dilated sulci for arteries and diploetic veins, often combined with enlargement of the foramen spinosum of the same side and perhaps in conjunction with structural changes of the theca cranii, are indicative of meningeoma of the convexity. Meningeoma of the sphenoidal ridge run their course with a characteristic thickening and sclerotisation of the bone. Memingeoma of the tuberculum sellae are characterised by hyperostosis of the tuberculum and meningeoma of the olfactory sulcus often give rise to hyperostosis and sclerosis of the olfactory sulcus and of the crista galli. Glioma of the chiasma as a rule gives rise to enlargement of the chiasmal sulcus. Glioma of the optic nerve as well as aneurysm of the opthalmic artery may cause the optic foramen to become enlarged. Adenoma of the hypophysis is characterised by 'ballooning' of the sella. Cranial pharyngeoma present typical calcifications and, like other suprasellar tumours, give rise to characteristic changes of the sellar entrance. Auditory neurinoma causes in well nigh 90 % of the cases a funnel-shaped enlargement of the internal auditory foramen and canal. The cases above mentioned are but a few instances of the information obtainable by skeletal radiographs as to the location and nature of a morbid process but do not pretend to make up a complete list.

Encephalography after cisternal puncture and inflation of air is the method of examination employed in the next place. Encephalography ought not to be used when there is suspicion of a tumor in the posterior fossa or when there is a marked increase of the intracranial pressure. An injection of 30 cc. of air is generally sufficient for producing the external subarachnoidal space with its cisterns as well as the different

contours of the cerebral ventricles. By a suitable technique the radiologist is able to do this by shifting the air to the various regions to be examined (LYSHOLM: *Das Ventrikulogramm*). The encephalogram may be said to delineate the outher contours of the brain as well as showing the topography of the ventricles and permits not only the study of expansive processes but also other morbid processes, such as atrophic conditions of the brain.

In cases where encephalography is contraindicated or if, on account of insufficient inflation of the ventricular system of both sides, the encephalogram does not permit of an exact diagnosis of the location of the cerebral tumour, then ventriculography is indicated. By performing the operation in immediate connection with the roentgen examination — with exstirpation of the tumour — or, where operation is contraindicated, by letting out cerebrospinal fluid and air through ventricular puncture we are able to keep the post-ventriculographic rise of pressure under control. By such means ventriculography is to be regarded as a practically harmless method of examination.

The encephalo- and ventriculograms are also of importance for diagnosing the precise nature of the tumour. In many cases we are able, by the former method to determine whether a convexity tumour is located within or outside the brain, of decisive importance for the differential diagnosis: convexity meningeoma — glioma. In most cases the ventriculogram also enables us to distinguish between meningeoma of the falx cerebri and glioma of the corpus callosum, a matter of the greater importance since the falx meningeoma on account of their cerebral blood supply produce no skeletal or vascular changes in the skul-cap. The corpus callosum glioma usually present an irregular outline against the air in the lateral ventricles which become forced apart by extension of the tumour into the septum pellucidum. Glioma and cysts of the septum pellucidum can also be diagnosed through the ventriculogram as well as intraventricular cholesteatoma, the latter presenting a characteristic picture against the air in the lateral ventricle. Even the nature of intraventricular meningeoma can be diagnosed if of sufficiently small size to be circumfluenced by the ventricular air. Malignant glioma invading the lateral ventricles can be determined as to their true nature thanks to their irregular outlines against the ventricular air and possible signs of invasion centrally or into the contra-lateral half of the brain. Cysts in the foramen Monroi and tumours arising in the choroid plexus further back in the roof of the third ventricle are revealed in the ventriculogram both as to their location and nature. New growths in the posterior part of the third ventricle showing a tendency to invade the central ganglia can be referred to the heterogeneous but from a surgical practical point of view

important group of inoperable malignant pinealoma. A depression of the proximal part of the aqueduct without deformity of the posterior portion of the third ventricle and without lateral displacement of the aqueduct or fourth ventricle is pathognomonic of glioma of the corpora quadrigemina. Glioma of the pons Varolii also yield a characteristic bowshaped dorsal displacement in the midline of the aqueduct and posterior part of the bottom of the third ventricle. The nature of small papilloma and ependymoma growing within the fourth ventricle and circumfluenced by the ventricular air can likewise be revealed in the ventriculogram. The possibility by ventriculography to determine the nature of a pathological process is further illustrated by the characteristic changes of the ventriculogram obtained in cases of stenotising ependymitis in the aqueduct and in arachnoiditis of the posterior fossa of the skull. In the former case we find a characteristic cornet-shaped stenosis of the aqueduct about 1 cm. below the third ventricle with a secondary internal hydrocephalus, usually most marked in the case of the third ventricle. Arachnoiditis of the posterior fossa of the skull produces a symmetrical internal hydrocephalus with well marked dilatation of the fourth ventricle.

It is in two different spheres of diagnostics that cerebral angiography is particularly indicated, vascular conditions and malformations which through arteriography alone can be studied as to their location and nature and then tumours, showing a vascularisation characteristic of the tumour in question, whereby its nature can be determined.

Arterio-venous aneurysms are malformations of congenital nature which usually but late give rise to symptoms in the form of subarachnoidal haemorrhages and epileptiform seizures, possibly combined with symptoms of cerebral inhibition. By arteriography such aneurysms can be mapped down prior to the operation with the view to establishing their in- and outlets, their distribution and location. They occur far more frequently than earlier believed and it is only after the introduction of arteriography that the possibilities of reliable diagnostics and thereby a rational surgical therapy have been opened to us.

Even the sack-shaped arterial aneurysms, usually localised to the intracranial portion of the internal carotid, the so-called carotid siphon, or to the art. comm. ant., are far more common than was formerly supposed to be the case. All according to their position they may give rise to typical infra- or supraclinoidal syndroms and are readily and unmistakably revealed by arteriography.

Lastly just a few words about the subdural haematoma. The arteriogram, taken in an antero-posterior projection, is here alone able to disclose the true nature of the condition by showing how the brain

surface, marked out by the end branches of the vessels, is forced inwards from the inner surface of the skul-cap.

The second large province of cases where arteriography is indicated concerns tumours with a characteristic form of vascularisation. As early as 1931 it was pointed out by MONIZ that richly vascularised tumours could be precisely localised by arteriography and that the nature of the tumour could be revealed by the appearance of this vascularisation. Since then MONIZ as well as LÖHR have given their conception of the characteristic arteriogram of meningeoma. They have also published cases of astrocytoma and oligodendroglioma, pinealoma and ependymoma, tumour metastases, and so on, with a vascularisation permeated and rendered visible by the opaque substance, in some cases in the form of arteries and veins filled with contrast, at other times in the form of a diffusely increased density of the tumour owing to its capillaries being filled with the contrast agent. The density of the tumour or the abundancy of the vessels filled with contrast can be said to indicate the degree of blood-flow through the tumour; from this, one has tried to draw certain conclusions regarding its nature. Apart from meningeoma, however, there has been no mention of any arteriograms, characteristic of different forms of tumour and enabling a closer study of their nature until TÖNNIS showed that the nature of glioblastoma could be revealed by arteriography.

TÖNNIS built his method on the observation that in glioblastoma the veins above and around the tumour carry a light-coloured arterial blood; he therefore concluded the presence of arteriovenous fistulae which one should be able to show by arteriography. He also proved this to be the case at the Surgical Congress in Magdeburg in 1936, thereby inaugurating the art of diagnosing the nature of malignant glioma by arteriography.

The indications and methods elaborated at the Neurological Clinic (Professor ANTONI), the Neuro-Surgical Clinic (Professor OLIVECRONA) and the Roentgenological Department (Dr. LYSHOLM) of the Seraphimer Hospital for the roentgenological examination of cerebral tumours may thus be briefly summarised as follows:

1) The flat-film examination of the skull, which affords a general orientation of the appearance of the brain cavity, which shows, in intracranial expansive processes with an increased pressure persisting for some time past, the consequent changes, and which also enables in some cases a definite diagnosis of the tumour present both regarding its location and nature.

2) Encephalography after cisternal puncture, which gives a general outline of the anatomical appearance of the brain and the topography

f the liquor spaces, and which permits a study of expansive processes s well as atrophic conditions of the brain.

3) Ventriculography for pre-operative exact localisation of a cere- ral tumour.

4) Arteriography for determining the location and nature of ar- rial aneurysms and vascular malformations as also malignant glioma.

Thanks to BAILEY and CUSHING, PENFIELD and others the glioma l ve been divided and classified into a number of groups, of which a l rge and important one has been labelled glioblastoma multiforme. As the name indicates, these tumours are characterised by their multi- f rm appearance, macro- as well as microscopically, and are built up of a number of immature glia cells with plenty of mitotic formations and n ultinuclear giant cells. They are further characterised by a tendency t haemorrhage and necrosis wherefore, macroscopically, they assume a n ottled brown-yellow appearance and present an uneven consistence with softer and more solid parts. Regarding their vascularisation BAI- LEY says in his publication, 'Intracranial Tumours': »Blood vessels are numerous and quite abnormal in structure. Their walls are thin and composed of an endothelial sheath surrounded by strands of collagenic tissue. There is never a muscular sheath. The connective tissue is usually degenerating, edematous, infiltrated with fat or hyalinized. Tortuosi- ties and aneurysmal dilatations are frequent. Proliferation of the in- tima and thromboses are numerous. From such thin-walled and dege- nerated vessels it is understandable that numerous haemorrhages and softenings occur.» In a recent work emanating from the Kaiser-Wil- helm-Institut für Hirnforschung ZÜLCH has made the following state- ment regarding the vascularisation of the glioblastoma: »In der nächsten Nachbarschaft und der Wachstumszone dieser Geschwülste finden wir auffällig weiträumige sinusartige Gefässe, die nach Beobachtungen bei der Operation anscheinend mit arteriellem Blut gefüllt sind. Sie wer- den von ganz dünnen Wänden mit zartem, venösem Bau eingesäumt.» Pathologically, anatomically and clinically the glioblastoma make up a fairly well defined form of tumour. They may be located anywhere in the cerebrum or brain stem and in the present material occur mostly between the age of 40 and 60. They are further characterised by their r pid and infiltrative growth, pay no heed to lobar boundaries and quick- ly attain a wide distribution. Clinically they are characterised by a brief h story of tumour, well marked psychical disturbances and often marked s gns of local brain defects.

Most neurological surgeons would seem to agree that, at any rate f r the present, these tumours are inaccessible for operative therapy w herefore it must be considered of very great value to be able to deter-

A. Verified malignant glioma with typical vascularisation	9			
B. Unverified » » » » »	14	23 cases	64 per cent	
C. Verified » » » atypical »	8	»	22	»
D. » » » without visible »	5	»		
Total: 36 cases				

Case	Sex	Age	Journ. (Cer. ang.)	Location	Verification
A. Verified with typical vascularisation					
1	M.	60 years	s. 52	centr. in thal.	Macr: Gl.bl.
2	M.	50 »	57	front	Pad: Gl.bl.
3	M.	40 »	63	occ.	»
4	M.	46 »	78	pa–front.	»
5	M.	60 »	80	front–centr.	»
6	M.	43 »	91	centr–gangl.	»
7	M.	60 »	92	temp.	»
8	F.	45 »	105	pa.	»
9	M.	64 »	108	temp–centr.	Macr: Gl.bl.
B. Unverified with typical vascularisation					
10	M.	39 years	s. 64	pa.	—
11	M.	53 »	70	front.	—
12	M.	47 »	74	pa.	—
13	M.	51 »	77	temp.	—
14	F.	46 »	79	temp.	—
15	F.	38 »	85	pa.	—
16	F.	50 »	93	temp.	—
17	M.	50 »	94	front–pa.	—
18	M.	41 »	95	pa.	—
19	M.	43 »	100	temp–occ.	—
20	M.	55 years	s. 102	temp.	—
21	M.	49 »	103	pa.	—
22	F.	57 »	110	pa.	—
23	M.	58 »	111	pa–occ.	—
C. Verified with atypical vascularisation					
24	M.	40 years	s. 21	temp.	Pad: Gl.bl.
25	M.	58 »	36	front.–c. callos	Pad: Astrobl.
26	M.	38 »	40	pa.	Pad: Gl.bl.
27	F.	53 »	51	temp–pa.	Macr: Gl.bl.
28	M.	42 »	67	pa.	Pad: Astrobl.–Gl.bl.
29	M.	61 »	82	temp–centr.	Pad: Gl.bl.
30	F.	52 »	99	temp–centr.	»
31	M.	51 »	n. 147	temp.	Pad: Astrocyt.–Gl.bl.
D. Verified without vascularisation					
32	F.	47 years	s. 23	temp.	Pad: Gl.bl.
33	M.	53 »	39	centr. in thal.	Macr: Gl.bl.
34	M.	54 »	n. 42	centr. in hypothal	Pad: Gl.bl.
35	F.	40 »	n. 46	front.	»
36	M.	50 »	s. 104	temp.	»

Abbreviations: Pad = Patho-anat. diagnosis; Macr = Macroscop. exam.; Gl.bl. = Glioblastoma; pa = parietally; front. = frontally; occ. = occipitally; centr. = centrally; temp. = temporally

ine preoperatively the nature of ese malignant glioma. The pati- t can thereby be spared a very ngerous operation associated ith a high primary mortality d which never can be radically rried out, nor does it usually sult even in a temporary return the working capacity.

The clinical material concerned i the present study is made up o 117 cases of arteriographed c rebral tumours which in the c urse of the last 5-year period, April 1934—May 1939, have been examined in the roentgenological department of the Seraphimer Hospital. These 117 cases also include arterial and arteriovenous aneurysms. 36 cases are made up of malignant glioma.[1]

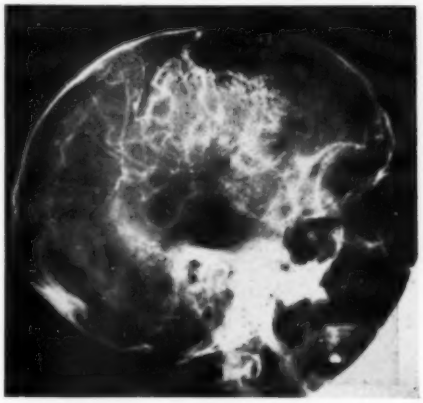


Fig. I. Case 4, male, aged 46. Journ. Cer. ang. s. 78. Headache for 4 months. Choked discs. Psychical derangement. Impaired speech. Arteriography: for malignant glioma typical vascularisation with opaque spots and arterio-venous fistulae. Efferent vein to sinus sagittalis. Autopsy: typical malignant glioma with haemorrhages and necrosis of the left parietal and frontal lobes. P. A. D. Glioblastoma.

The arteriographic appearance of the malignant glioma is in our experience characterised by an abundancy of new vessels within the tumour showing quite an irregular course and varying calibres. Between the vessels there are irregular opaque spots of smaller size which must be interpreted as contrast in miliary aneurysms (Cf. BAILEY). In addition arterio-venous fistulae are very often to be seen with contrast filling of one or more veins leaving the tumour and visible even on the arteriogram. This arteriogram which not only verifies the clinical observation of arterio-venous fistulae but also corresponds with the patho-anatomical picture of the vascularisation of the malignant glioma will in what follows be considered as *typical* for malignant glioma. We have never seen it associated with any other type of tumour than malignant glioma and, judging from the literature, nor has any other author. In our opinion therefore, it is pathognomonic of glioblastoma. (Fig. I.)

Our material of malignant glioma will be summarised in the opposite table.

[1] My heartful thanks are due to Prof. OLIVECRONA (chief for the Neuro-surgical Clinic) and Prof. ANTONI (chief for the Neurological Clinic) for permitting me to use the clinical data concerned.

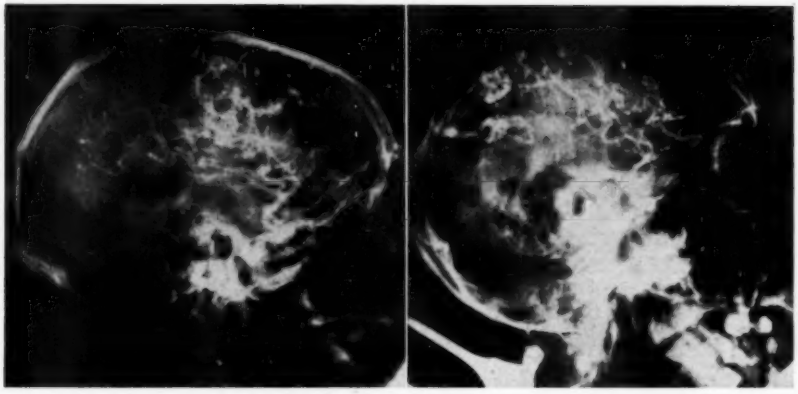


Fig. II. Case 15, female, aged 38. Journ. Cer. ang. s. 85. Headache for one month. Vomiting and impaired speech. Choked discs. Psychical derangement.

Observe the opaque veins emptying into the superior longitudinal sinus which also seems to be faintly filled with contrast.

Fig. III. Case 3, male, aged 40. Journ. Cer. ang. s. 63. Headache for the past 6 months in addition to mental derangement. Choked discs. Sensory and motor aphasia. Extract from surgical report: »Removal of large tumour widely adherent to the dura and quite indistinctly bounded from the surroundings. The veins adjacent to the tumour were much dilated, up to the width of a quill, and contained arterial blood.»

P. A. D. Glioblastoma.

Note the wide opaque veins in the arteriogram and the contrast in the sup. longitudinal sinus.

By macro- or microscopic examination of the tumour 22 cases were verified. In addition there is a group of 14 cases, all malignant glioma clinically established, but where operative or autopsic verification is lacking. In all these cases the arteriogram was the typical one for malignant glioma and the clinical diagnosis in all the 36 cases was likewise malignant glioma.

As has already been mentioned arterio-venous fistulae constitute one of the characteristic signs of malignant glioma. That fistulae of that nature are really present seems to us to have been proved by the fact that even the radiograms taken during the first phase of the opaque injection give evidence of contrast in vessels coming from the tumour and emptying into the sinus, and this a long time before other veins in the brain appear filled with the contrast agent. (Figs. II and III.)

In some cases pathologic vessels are only seen in the periphery of the tumour. This can be explained partly by extensive disintegrating necrotic foci in the centre of the tumour, partly by a more abundant va-

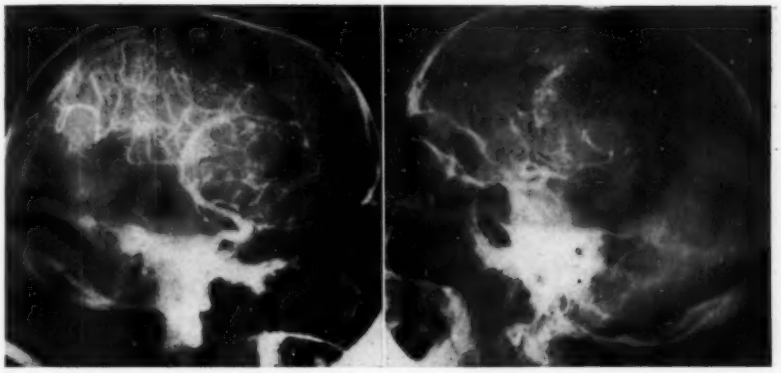


Fig. IV. Case 11, male, aged 53. Journ. Cer. ang. s. 70. For the past 3 weeks headache and mental disturbance. Choked discs. Dull and apathetic. Memory and power of concentration impaired, reaction time prolonged. Sensory aphasia with alexia and agraphia.

Note: irregular vessels and contrast patches in the periphery of the tumour. No arterio-venous fistulae visible.

Fig. V. Case 12, male, aged 47. Journ. Cer. ang. s. 74. For the past 7 months been having epileptiform seizures and severe mental disturbances.

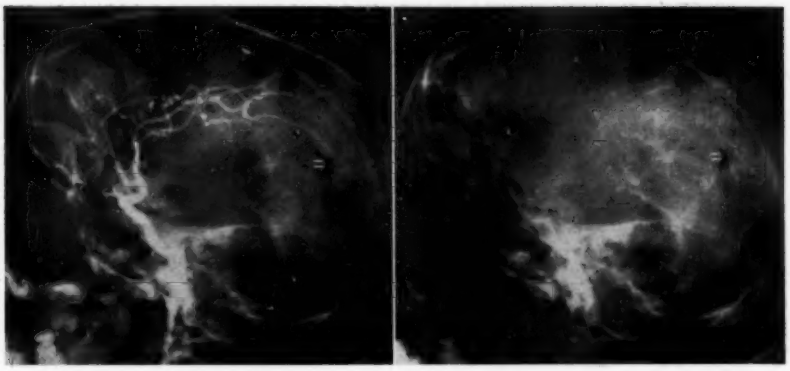
Note: irregular vessels of various sizes and contrast patches in the periphery of the tumour. Opaque vein passing up towards the sinus sagittalis.

cularisation in the bounding area between the tumour and surrounding cerebral tissue Cf. Zülch. (Figs. IV and V.)

The opaque filling of the vessels of the tumour appears as a rule best in the arteriogram but in three cases the vascularisation of the tumour has been best observed in the phlebogram. (Figs. VI and VII.)

In practically all the cases there remains in the phlebogram a faint opaque filling of the capillaries of the tumour in the form of a more diffuse patch of contrast; within and around this faintly opaque veins can be seen. And this after all the other arteries and capillaries have emptied themselves and the opaque substance has passed out into veins and the sinus which shows that the blood permeation of malignant glioma is delayed in relation to other healthy parts of the brain.

In addition to the visible vascularisation of the tumour we find the displacements of surrounding vessels and vascular groups usual for expansive processes. Thus we find the carotid siphon and sylvian group displaced downwards in a parietal position of the tumour, forwards-upwards in a temporal position, the cerebri anterior displaced toward the healthy side in a fronto-lateral and temporal position of the tumour and so on. Arteries passing to the tumour show no decided hypertrophy. The circulatory intravasal pressure in malignant glioma seems to be



a. b.

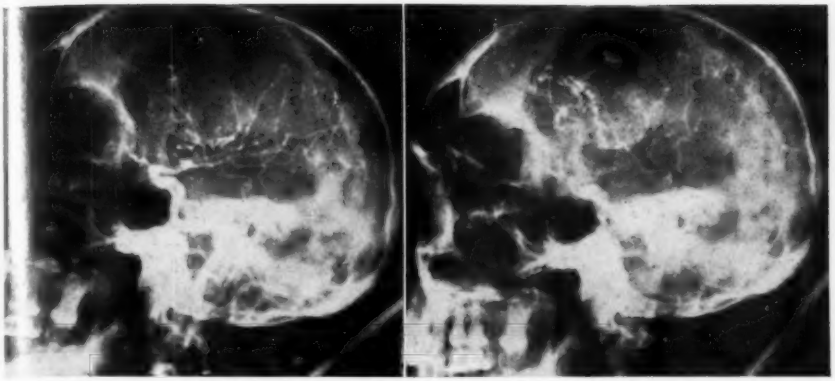
Fig. VI. Case 7, male, aged 60. Journ. Cer. ang. s. 92. Six months' history of epileptiform fits, headache, impaired memory and mental deterioration. Choked discs. Autopsy: in the posterior part of the temporal lobe a malignant glioma with haemorrhages and foci of necrosis.
P. A. D. Glioblastoma. Cf. arteriogram and phlebogram.

fairly normal. In but 3 out of our 36 cases was the pericallosal artery not filled with contrast, and the posterior cerebral artery was filled up in 10 cases.

So far as the differential diagnosis is concerned it is only the arterio-venous aneurysm that might be mistaken for the typical arteriogram of the malignant glioma. The aneurysm however does not present any definite expansive character with displacement of other vessels and vascular groups. The aneurysm has coarse hypertrophic afferent arteries and wide open fistulae between afferent and efferent vessels. In aneurysm of the a. cerebri media-region the pericallosal artery is not therefore usually filled, the aneurysm drawing all the blood as a sponge from the internal carotid of the affected side. On account of the rapid blood-flow occasioned by the wide arterio-venous fistulae the contrast in the phlebogram has already left the aneurysm and reached veins and sinus. On all these points the glioblastoma and the arterio-venous aneurysm show diametrically opposite conditions. (Figs. VIII and IX.)

As evident from the table the verified cases of malignant glioma include a group, C, of 8 cases where the vascularisation is denoted as *atypical*. In these cases the tumour shows a more regular hypervascularisation with the vessels arranged in a streak- or broom-shaped manner without any visible patches of contrast or arterio-venous aneurysms. (Fig. X.)

In two of these cases, too, the vascularisation is most marked towards the periphery of the tumour. (Fig. XI.)

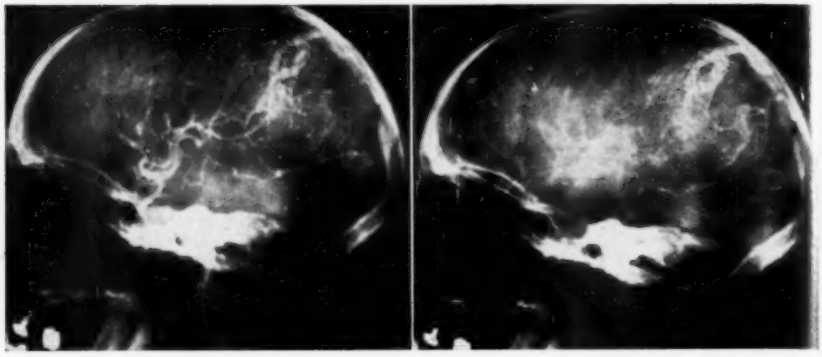


a. b.

Fig. VII. Case 17, male, aged 50. Journ. Cer. ang. s. 94. Headache for 12 months, the last 2 months mental derangement. Choked discs. Paresis of left arm and leg. Well marked dementation. Cf. arteriogram and phlebogram.

Among these cases with atypical vascularisation the histological diagnosis is in 5 cases glioblastoma, in 1 case astroblastoma on the way becoming glioblastoma, in 1 case astrocytoma on the way becoming glioblastoma and in 1 case astroblastoma. In nearly half the number of cases in this group the histological examination therefore does not show such a primitive and immature form of tumour as in the cases belonging to group A. There is thus a noteworthy parallelism here between the histological appearance and the arteriogram, in that among three cases it was only a glioma bordering on malignancy that showed a more regular hypervascularisation than that typical for malignant glioma.

Apart from these cases of *typical* and *atypical* vascularisation in malignant glioma we have not come across any visible vascularisation in our cases of arteriographed cerebral tumours except in two cases of meningeoma and metastatic tumours which have shown a vascularisation different from that of malignant glioma and which will be accounted for later. Our clinical material also includes 23 cases of benign glioma, of which 8 were astrocytoma, 6 oligodendroglioma, 7 unclassified and 2 ependymoma. In none of these cases does the arteriogram show any vascularisation of the tumour. The atypical vascularisation must therefore according to our experience cause one to suspect the tumour of being malignant. Yet there are cases published of astrocytoma as well as other benign gliomas where the tumour shows an atypical streakshaped vascularisation similar to that dealt with here.



a. b.

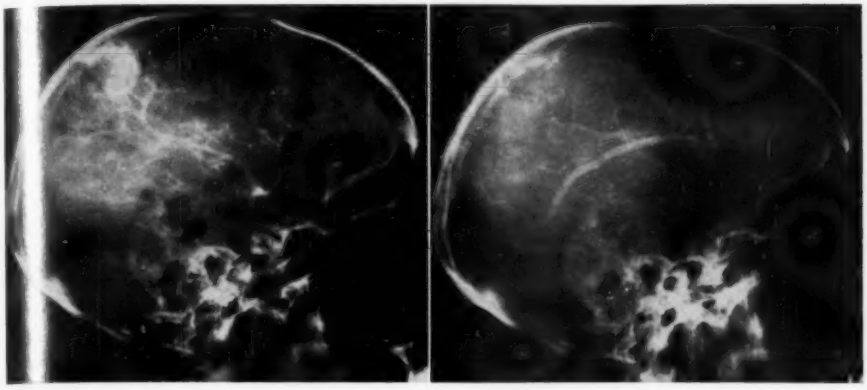
Fig. VIII. Case 8, female, aged 45. Journ. Cer. ang. s. 105. During the last month severe headache and mental disturbances. Choked discs. Extract from surgical report: »..... tumour adherent to the dura over an area the size of a child's hand..... macroscopically well localised but in a few places it had infiltrated the brain. On either side of the tumour there were two veins, as wide as quills, containing arterial blood..... The entire tumour was enucleated with the finger.»

P. A. D. Glioblastoma.

Note the forcing apart and displacement of the end arteries of the sylvian group. No hypertrophy of afferent arteries. Pericallosal artery filled with contrast. Contrast in the capillaries and veins is seen in the phlebogram.

Already mentioned, however, we have not been able to verify this observation.

Of the malignant gliomas there finally remains a small group, D, of 5 cases which do not show any visible vascularisation of the tumour. Two of these are located centrally and if their nutrition is mainly supplied by the slender aa. striae-thalamici from the middle cerebral artery, which normally only in exceptional cases become filled with contrast, this may explain why the vascularisation of the tumour fails to appear in the arteriogram. Compare however group A where two malignant gliomas, centrally situated, were obtained filled with contrast. Regarding the other 3 cases in this group one is located frontally and 2 temporally. In one of the two latter cases the posterior cerebral artery is filled but not in the other. As to the frontal case the tumour here was located in the middle of the frontal lobe and was adherent to the falx cerebri. In these cases the afferent arteries must presumably be thrombosed whereby the contrast has failed to reach the interior of the tumour. The same explanation can of course be applied to the two centrally situated malignant gliomas of this group. Neither autop-



a. b.

Fig. IX. Female, aged 35. Journ. Cer. ang. s. 114. Clinical diagnosis: Cerebral tumour. Arterio-venous aneurysm verfified. Subarachnoidal haemorrhage 3 years ago. Since then another heaemorrhage and epileptic fits. Operation: removal of a typical arteriovenous aneurysm from the left parietal region.

P. A. D. arterio-venous aneurysm.

Note that there is no displacement of the sylvian artery which is coarse and hypertrophic. Wide fistulae between afferent and efferent vessels. Pericallosal artery not filled with contrast. No contrast in the aneurysm is seen in the phlebogram.

nor operative reports give any information in this respect. In none of the cases is there any definite necrotisation of the tumour.

In summary it may be said of the malignant gliomas that in about 64 % of our material they showed a vascularisation pathognomonic of glioblastoma by which the nature of the tumour could be definitely diagnosed. In another 22 % pathological vessels appeared in the arteriogram which, even if permitting of no definite diagnosis as to the nature of the tumour, at any rate showed the picture of a vascular tumour liable to raise the suspicion of a malignant glioma or one bordering on malignancy. In a total of 86 % the visible vascularisation of the malignant glioma enabled its exact localisation through arteriography alone.

According to MONIZ and LÖHR the meningeoma also appear in a large number of cases in the arteriograms. They are characterised, according to MONIZ, of fine vascular bundles ending in tiny black spots in which the vascular design disappears. In suspected meningeoma, LÖHR, like always MONIZ, carries out the contrast injection into the common carotid artery and in so doing he generally finds the meningeoma in the arteriogram. Our material of arteriographed cerebral tu-

Fig. X. Case 30, female, aged 52. Journ. Cer. ang. s. 99. For the past 2 months been having severe headache and Jacksonian epileptic fits. Incipient papilledema. Aphasia. Right-sided hemiparesis. Mentally deranged. Autopsy: large malignant glioma in the left temporal lobe invading the internal capsule and basal ganglia. The tumour is permeated by necrotic and haemorrhagic foci.

P. A. D. glioblastoma.

Note: Atypical vascularisation without blotches or arterio-venous fistulae.

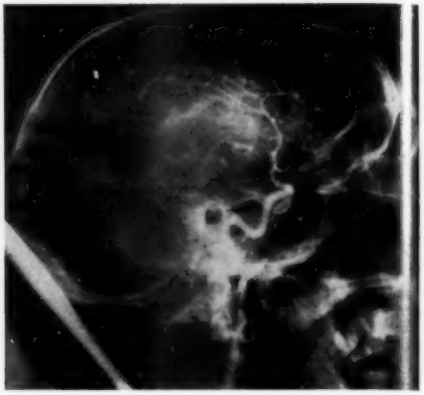


Fig. XI. Case 29, male, aged 61. Journ. Cer. ang. s. 82. For the past 6 months headache and mental disturbances. Choked discs. Autopsy: tumour of a malignant glioma type, about the size of a tangerine, permeated by numerous haemorrhages and infiltrating large parts of the left temporal lobe and extending deeply into the brain, causing destruction to the nucleus lentiformis etc.

P. A. D. Glioblastoma multiforme.

Note finely streaked vascularisation of the tumour, mainly peripherally.

mours include 10 cases of meningeoma of which two only appear with a visible vascularisation in the roentgen picture. This low figure may be due to the fact that the opaque injection in all the cases was carried out into the internal carotid. The meningeoma appearing in the arteriogram are a frontal convexity meningeoma and an olfactory meningeoma, both verified through histological examination after exstirpation. The latter tumour seems to have been filled via the ophthalmic artery, the former through collaterals from the anterior cerebral artery. In both our cases the meningeoma appears as a more diffuse, not fully homogeneous opaque spot, most clearly in evidence in the phlebogram. Its arteriographic appearance differs decidedly from that of the malignant glioma. (Fig. XII.)

The convexity meningeoma is seen in the arteriogram to be surrounded by circular arteries running round the tumour. The same picture is obtained in another case of convexity menigeoma, showing the

a. b.

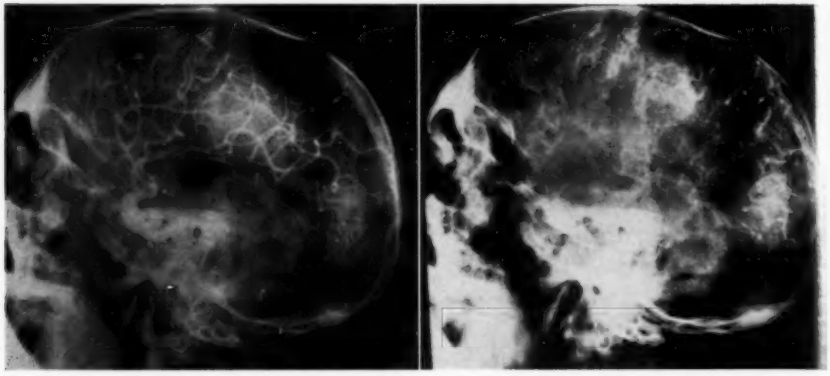
Fig. XII. Male, aged 52. Journ. Cer. ang. s. 96. Clinical diagnosis: Cerebral tumour; verified convexity meningeoma. Headache for 4 months. Choked discs. Operation — tumour removed.
P. A. D. Meningeoma.
Note on the phlebogram the fairly homogeneous contrast coloration of the tumour.

location of the tumour, but the tumour itself here does not present any definite coloration of the contrast agent. It would seem a rational procedure in meningeomas to inject the contrast agent into the common carotid since the nutrition of the meningeal tumours is usually mainly obtained from the external carotid via the middle meningeal artery.

Of cerebral metastatic tumours we have arteriographed 6, of which 5 were cancer metastases and 1 a metastatic melanosarcoma, all verified by histological examination. Two of these 6 cases, both cancer metastases, show a characteristic appearance in the phlebogram. In the arteriograms there is but a faint indication of a diffuse contrast coloration of the tumour, in the phlebograms there are annularly arranged, exceedingly slender irregular vessels in the periphery of the tumour, apparently corresponding to vessels in the tumour capsule. (Fig. XIII.)

The remaining 4 cases of metastatic tumours show no definite vascularisation of the tumour.

The material presented here is too small (perhaps with the exception of the malignant glioma) to permit of any far-reaching conclusions. The glioblastomas however showed in 64 % a characteristic and pathognomonic arteriogram. In still another 22 % they presented a more atypical hypervascularisation indicating the presence of a vascular tumour which, according to our experience, should raise the suspicion of malignant glioma. Our cases of benign glioma showed no visible vascu-



a. b.

Fig. XIII. Male, aged 58. Journ. Cer. ang. s. 71. Clinical diagnosis: Cerebral tumour; verified multiple cancer metastases.

For the past 12 months mental dulling, headache and choked discs. Left-sided hemiparesis. Autopsy: numerous secondary deposits of a size from peas to barely that of plums. P. A. D.: squamous cell epithelioma.

Note the phlebogram with annularly arranged contrast-filled slender vessels in the tumour capsule. In the arteriogram there is only a slightly bow-shaped basal displacement of the posterior part of the pericallosal artery.

larisation of the tumour. Meningeomas and metastatic tumours seem to show, in so far they appear in the arteriogram, arteriographic pictures differing from that of malignant glioma.

Further observations and investigations of a large number of carefully examined and verified cases will have to show whether it will be possible by cerebral angiography to elaborate still further the differential diagnosis concerning the precise nature of various cerebral tumours.

SUMMARY

After a brief account of the possibilities afforded by the flat film examination of the skull and the ventriculogram for diagnosing the nature of intracranial expansive processes and after mentioning the importance of arteriography for the diagnosis of vascular diseases and malformations the author describes the arteriographic appearance of malignant glioma as presented in 36 cases of such nature. In 23 cases (64 %) the arteriogram offered a characteristic picture with an abundancy of newly formed pathological vessels with miliary aneurysms and arterio-venous fistulae. This arteriogram verifies the clinical observation of arterio-venous fistulae and corresponds to the patho-anatomical picture of the vascularisation of the malignant glioma and is denoted as pathognomonic of malignant glioma. Stress is laid on the great value of preoperative diagnosis of the nature of malignant glioma, inaccessible to surgical therapy. The differential diagnosis from arterio-venous aneurysm is discussed. Out of the remaining 13 cas

arteriographed malignant glioma 8 showed a more regular hypervascularisation without any aneurysmatic patches or arteriovenous fistulae. In the author's experience a vascularisation of that nature should also raise the suspicion of malignancy of the tumour as all the arteriographed cases of benign glioma, 23 in all, failed to show any visible vascularisation of the tumour. 5 cases of malignant glioma showed no pathologic vessels in the arteriogram.

The author finally records 2 cases of meningeoma and 2 cases of metastatic cancer showing a vascularisation that differed from one another as well as from that of malignant glioma.

ZUSAMMENFASSUNG

Nach einem kurzgefassten Bericht über die Möglichkeiten, die die röntgenologische Schädeluntersuchung und das Ventrikulogramm für die Artdiagnostik intrakranialer expansiver Prozesse bietet, und nach Erwähnung der Bedeutung der Arteriographie für die Diagnostik vaskulärer Krankheiten und Missbildungen beschreibt Verf. das arteriographische Bild des malignen Glioms, wie es bei 36 Fällen von arteriographierten malignen Gliomen hervorgetreten war. In 23 Fällen (64 %) bot das Arteriogramm ein charakteristisches Bild mit grossem Reichtum an neugebildeten pathologischen Gefässen mit miliären Aneurysmen und arterio-venösen Fisteln. Dieses Arteriogramm verifiziert die klinische Beobachtung arterio-venöser Fisteln, entspricht dem pathologisch-anatomischen Bilde der Vaskularisierung des malignen Glioms und wird als pathognomonisches Merkmal des malignen Glioms bezeichnet. Verf. betont den grossen Wert einer präoperativen Artdiagnose maligner Gliome, die der chirurgischen Therapie unzugänglich sind, und erörtert die Differentialdiagnose gegen arteriovenöses Aneurysma. Von den übrigen 13 Fällen von arteriographierten malignen Gliomen zeigten 8 eine regelmässigere Hypervaskularisierung ohne aneurysmatische Flecken oder arteriovenöse Fisteln. Nach der Erfahrung des Verfassers soll auch eine solche Vaskularisierung Verdacht auf Malignität des Tumors erwecken, da sämtliche arteriographierte Fälle von benignen Gliomen, 23 an der Zahl, keine sichtbare Vaskularisierung des Tumors aufwiesen. 5 maligne Gliome zeigten keine pathologischen Gefässe auf dem Arteriogramm.

Schliesslich teilt Verf. 2 Fälle von Meningeom und 2 Fälle von Karzinommetastasen mit, die eine voneinander und von den malignen Gliomen abweichende Vaskularisierung zeigten.

RÉSUMÉ

Après un bref exposé des possibilités que l'examination radiographique du crâne et les ventriculogrammes présentent dans le diagnostic d'espèce des foyers expansifs intracraniens, ainsi qu'après avoir mentionné l'importance de l'artériographie pour le diagnostic des maladies vasculaires et malformations, l'auteur décrit l'image artériographique du gliome malin, telle qu'elle ressort de 36 cas de gliome malin artériographiés. Dans 23 cas (64 %) l'artériogramme offrait une image caractéristique avec une grande richesse de vaisseaux néoformés pathologiques, pourvus d'anévrysmes miliaires et de fistules artério-veineuses. Cet artériogramme vérifie l'observation clinique de fistules artério-veineuses et correspond à l'image anatomo-pathologique de la vascularisation du gliome malin et est déclaré pathognomonique du gliome malin. Là grande importance d'un diagnostique d'espèce, préopératif, des gliomes malins, qui sont inaccessibles à la thérapeutique chirurgicale, est soulignée. Le diagnostic différentiel des anévrysmes artério-veineux est discuté. Sur les 13 autres cas de gliome malin artério-

graphiés 8 présentèrent une hypervascularisation plus régulière, sans taches anévrismatiques et sans fistules artério-veineuses. Selon l'expérience de l'auteur même une telle vascularisation doit faire supçonner une tumeur maligne, puisque sur les 20 cas artériographiés de gliome bénin aucun ne présentait une vascularisation visible de la tumeur. 5 gliomes malins ne montraient pas de vaisseaux pathologiques sur l'artériogramme.

Enfin 2 cas de méningéome et 2 cas de métastases cancéreuses sont indiqués. Ils présentaient une vascularisation différente entre eux et différente de celles des gliomes malins.

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III. An Account of the Second International Congress of Radiology, Stockholm, 1928. Price Sw. cr. 15: —.

IV. Teaching and Training in Medical Radiology. Papers delivered at the Second International Congress of Radiology. Price Sw. cr. 20: —.

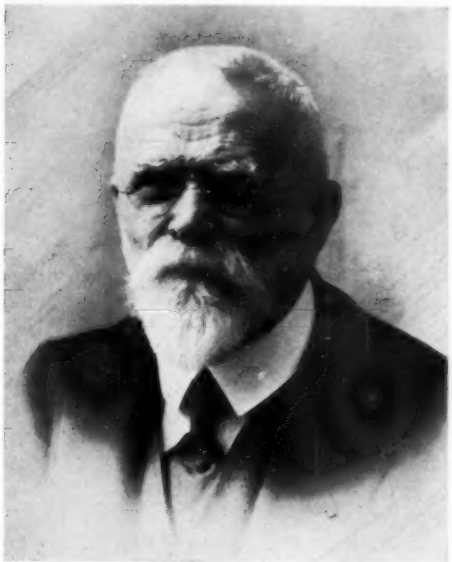
V. Catalogue des Portraits des Membres du Deuxième Congrès International de Radiologie à Stockholm. 23—27 Juillet 1928. 2ème Edition, complétée. Price Sw. cr. 5: —.

VI. Gunnlaugur Claessen: *The Roentgen Diagnosis of Echinococcus Tumors.* Price Sw. cr. 15: —.

VII. Martin Odin & Gösta Runström: *Iodized Oils as an Aid to the Diagnosis of Lesions of the Spinal Cord and a Contribution to the Knowledge of Adhesive Circumscribed Meningitis.* Price Sw. cr. 15: —.

VIII. Carl Krebs: *The Effect of Roentgen Irradiation on the Interrelation between Malignant Tumors and their Hosts.* Price Sw. cr. 8: —.

IX. Jens Juul: *Experimental Studies on Roentgen Treatment of Malignant Tumors.* Price Sw. cr. 8: —.



ANTOINE BÉCLÈRE

* 17. III. 1856 † 24. II. 1939

In Memoriam

ANTOINE BÉCLÈRE, le grand pionnier français de la radiologie médicale, est décédé subitement, le 24 février 1939, en son domicile, 122 Rue La Boëtie à Paris, d'une crise cardiaque.

Dans le magnifique »Livre Jubilaire» qui fut offert au docteur ANTOINE BÉCLÈRE à l'occasion de son 80e anniversaire, en 1936, le professeur CL. REGAUD a présenté la carrière et l'œuvre scientifique du maître dans son ensemble, et treize de ses élèves y ont rédigé des exposés sur les différentes branches de son vaste œuvre. Le Livre Jubilaire contient aussi une bibliographie et un index chronologique des publications d'ANTOINE BÉCLÈRE jusqu'à 1936, se montant au nombre de 242.

Dans les »Fortschritte a. d. Geb. d. Rö. strahlen», Vol. LIX N:o 6, Mlle ANTOINETTE BÉCLÈRE a donné un beau nécrologe de son illustre père. Elle y dit, avec une fierté bien fondée: »Depuis ses 80 ans il avait encore ajouté 20 publications nouvelles à la liste de ses nombreux travaux».

Le présent article veut donner aux lecteurs de ce journal un court exposé des aspects du grand maître et de son œuvre qui, à mon avis, ont eu la plus grande influence sur le développement de la radiologie.

Pour bien comprendre le rôle de pionnier de BÉCLÈRE dans le domaine de la rœntgenologie, il faut avoir été témoin de l'effet causé par la découverte de ROENTGEN à la fin du siècle dernier dans le monde de la médecine. Il faut nous rappeler sous quelles conditions travaillaient les grands pionniers de la roentgenologie.

Quand, en février 1896, C. W. ROENTGEN démontra la radiographie des os de la main humaine en faisant ressortir l'importance de la radiographie pour la médecine, il posa ainsi les fondements du roentgendiagnostic. Peu après on fut à même d'observer sur l'écran fluorescent les mouvements du cœur et du diaphragme et on commença à étudier la forme et les mouvements du canal digestif à l'aide de préparations de sous-nitrate de bismuth. Dans l'espace d'une année, à compter du jour de la découverte de ROENTGEN, on installa un peu partout, dans le monde, de petits cabinets de radiographie dans les caves et lieux de débarras. On y étudia les radiographies de différents états pathologiques avec la ferveur et la vive curiosité de l'homme lors de l'exploration de champs nouveaux dans le domaine de la science.

La roentgenthérapie est née dans la même année que le roentgendiagnostic. Il est moins facile de comprendre que l'on put découvrir si vite l'effet thérapique des rayons de ROENTGEN. Mais le terrain était préparé pour la roentgenthérapie par la découverte de l'héliothérapie artificielle.

L'ophtalmologue suédois JOHAN WIDMARK avait en 1889 découvert et décrit que les rayons ultra-violets du spectre solaire possédaient l'effet biologique le plus prononcé et que c'était ces rayons qui lors d'une action intense des rayons solaires produisaient une inflammation aiguë dans la peau. Ensuite le danois NIELS R. FINSEN, le créateur de la finsenthérapie, avait démontré, dans une série d'ouvrages publiés au cours des années suivantes, qu'une réaction de la peau causée par des rayons ultra-violets était à même de produire la guérison de la tuberculose et du cancer de la peau. Il semble tout naturel que les dermatologues, qui connaissaient les découvertes de FINSEN, aient alors voulu étudier si la réaction que produisaient les rayons de ROENTGEN dans la peau lors d'une irradiation intense pouvait avoir un effet curatif. En effet, au début, on croyait même généralement que, dans la roentgenthérapie, c'était des rayons ultra-violets émanant du tube de roentgen qui produisaient l'effet curatif.

Après que les médecins viennois FREUND, SCHIFF et KIENBÖCK eurent, en 1896 déjà, décrit des résultats favorables obtenus par l'irradiation de Roentgen sur des dermatomycoses, dermatoses, dermatites et tuber-

ses de la peau, on commença à étudier, dans le monde entier, l'effet s rayons de Roentgen sur des maladies de toutes sortes. Déjà en 1896, l français DESPEIGNES prétendit avoir constaté une régression, après r ntgenthérapie, d'un cancer inopérable de l'estomac, et l'allemand OCHT décrivit en 1897 une amélioration subjective passagère de quel- es cas de cancer inopérable du sein. En 1900, les suédois STENBECK SJÖGREN communiquèrent qu'ils avaient réussi à guérir des cas de ncer de la peau par les rayons de roentgen. En 1901, l'américain RL BECK constatait la disparition d'un mélanosarcome après un même itement. La même année, FRANCIS H. WILLIAMS de Boston décrivit ux cas de cancer des lèvres et, en 1903, six cas de métastases cutanées cancer du sein et un cas de cancer mammaire primaire chez un mme, lesquels il avait réussi à faire disparaître par la roentgenthérapie.

Déjà à la fin du siècle dernier, la roentgenthérapie commençait à se re une place aussi pour le traitement de maladies se rapportant à médecine interne. En 1898, l'allemand H. RIEDER publia un mémoire venu classique sur l'effet de la radiation de roentgen sur les microbes sur le mécanisme de guérison d'états inflammatoires bactériogènes.

En 1900, STENBECK donna communication de 52 cas de rhumatisme articulaire où, dans 40 % des cas, il avait pu constater une amélioration subjective et objective. En 1901, CARL BECK annonça une amélioration d'un goître traité par les rayons de roentgen et proposait l'irradiation de la glande thyréoïdienne dans la maladie de Basedow.

A la même époque, CARL BECK et SENN de New York firent sensation en annonçant qu'ils avaient obtenu une amélioration de longue durée et même une disparition des symptômes de la leucémie et de la pseudo-leucémie par l'irradiation de la rate et des infiltrations glandulaires; WILLIAMS, de même, en démontrant qu'il avait pu guérir des lymph-adénites tuberculeuses, et PUSEY en communiquant qu'il avait réussi à faire disparaître des tumeurs glandulaires dans la maladie de Hodgkins après roentgenthérapie. Plusieurs auteurs parlaient déjà à cette époque de l'effet analgésique des rayons de roentgen.

On peut se rendre compte du développement de la roentgenthérapie en constatant que FRANCIS H. WILLIAMS de Boston pouvait, lorsqu'il publia en 1903 la 3ème édition de son célèbre manuel «The Roentgen Rays in Medicine and Surgery», en se basant sur son expérience personnelle et sur la littérature, présenter des résultats positifs obtenus par l'irradiation de roentgen dans presque tous les domaines de la médecine qui à l'heure actuelle sont justiciables de la roentgenthérapie.

Au début, la pénétration des rayons de Roentgen dans le domaine de la médecine causa avant tout de l'étonnement et excita la curiosité armi les médecins praticiens. Plusieurs accueillirent les communications es découvertes roentgenologiques avec scepticisme et même s'en moquè-

rent. Mais en peu de temps, le nombre augmenta de ceux qui comprirent que, par les rayons de roentgen, la science médicale avait obtenu une arme nouvelle ouvrant de nouveaux champs d'activité au diagnostic et à la thérapie.

Les revues médicales furent encombrées par des communications sur »l'image roentgenologique» de différents états pathologiques et sur l'effet thérapique des nouveaux rayons que l'on avait pu constater. Et déjà au cours des années qui suivirent immédiatement la découverte de ROENTGEN les premières revues spéciales pour la roentgenologie médicale furent fondées en Angleterre, en Allemagne et aux Etats-Unis. La France possédait en les »Archives d'Electricité Médicale», rédigées par J. BERGONIÉ, un journal scientifique qui, dès le début, publia les innovations dans le domaine de la roentgenologie, et pendant les premières dix années de ce siècle FOVEAU DE COURMELLES publia annuellement un volume de son ouvrage vivace et érudit »L'Année Electrique, Electrothérapique et Radiographique» qui, pour les roentgenologistes de ce temps, représentait un regard ouvert vers le monde.

Un grand fonds de connaissances fut ainsi vite accumulé sur l'emploi de la roentgenologie dans différents domaines de la médecine, mais il s'écoula plusieurs années avant que ces connaissances dispersées fussent réunies pour former une science ordonnée.

Pour les médecins qui à cette époque furent captivés par les vastes horizons qu'offrait la nouvelle science, c'était un grand problème que d'obtenir un enseignement organisé de toutes ces nouvelles observations, une instruction compétente de cette nouvelle technique inconnue et de l'interprétation de ces ombres de la réalité. Pour chaque terrain conquis sur le champ roentgenologique, de nouvelles sources d'erreurs et de nouveaux dangers semblaient se présenter.

Celui qui a assisté soi-même à ces évènements n'oubliera jamais le sentiment particulier de fascination et en même temps d'impuissance qu'éprouvait le jeune médecin devant ce chaos d'innovations. Où pourrait-il trouver un professeur capable de donner un exposé critique et systématique des expériences roentgenologiques, un maître qui possédât à fond aussi bien la technique que la matière scientifique, un phare pouvant le guider à travers tous les écueils?

Celui qui au commencement de ce siècle voulait s'instruire dans le domaine de la roentgenologie apprit assez vite qu'il n'existait qu'un petit nombre de phares pouvant lui montrer le chemin sur les vastes espaces de ce domaine. ANTOINE BÉCLÈRE de Paris était un de ces illustres maîtres pionniers.

ANTOINE BÉCLÈRE naquit à Paris le 17 mars 1856. Sa mère était du Mâconnais. Son père, le docteur CLAUDE BÉCLÈRE, médecin de quartier à Paris, était d'une famille de cultivateurs de la Bresse. Il fit ses études

au Lycée Condorcet où il était dans les premiers. Il prit son inscription à la Faculté de Médecine à la fin de 1873 et entra comme bénévole chez le Professeur TILLAUX. Externe des Hôpitaux en 1875, il en est nommé Interne en 1877. Il ambitionnait de faire de la médecine proprement dite mais se spécialisa par occasion dans l'ophtalmologie et l'otologie. Ainsi au service de chirurgie générale, DUPLAY chargea le jeune BÉCLÈRE d'une consultation d'otologie et d'une autre d'ophtalmologie. BÉCLÈRE fut finalement conquis par la pédiatrie. Vivement intéressé par les questions de pathologie générale relatives à la contagion et à l'immunité il fit en 1882, alors interne à l'Hôpital des Enfants-Malades, sa thèse, sous la direction de son maître LABRIC, sur »La contagion de la rougeole».

En 1893, BÉCLÈRE fut nommé médecin des Hôpitaux et ses premiers services furent à l'Hôpital St. Louis, l'Hôpital du Bastion 29 et l'Hospice Debrousse. Au début de 1897, il devint médecin de l'Hôpital Tenon où il créa immédiatement, à ses frais, un laboratoire de roentgendiagnostic. A la fin de 1897, il passa à l'Hôpital St. Antoine où il travailla jusqu'en 1921 quand il fut atteint par la limite d'âge.

A l'Hôpital St. Antoine une petite chambre à l'entrée de son service fut mise à sa disposition pour servir de laboratoire radioscopique. Pour la radiographie, les patients furent au commencement conduits de l'hôpital au domicile de BÉCLÈRE.

L'activité scientifique de BÉCLÈRE fut partagée entre la médecine générale et la roentgenologie. Environ trois quarts de ses travaux sont consacrés à la roentgenologie, au diagnostic et à la thérapie à peu près dans les mêmes proportions. Le reste se réfère à l'épidémiologie, la sérologie et l'endocrinologie.

Quand, au début de 1896, BÉCLÈRE commença à étudier la roentgenologie, il était déjà un auteur renommé dans le domaine de l'immunité et de l'opothérapie. Il continua sa production scientifique dans la médecine générale, à côté de son œuvre roentgenologique, jusqu'en 1900. A partir de 1901 il consacra ses recherches pendant trois dizaines d'années exclusivement à la science roentgenologique, pour reprendre enfin pendant ses dernières années, avec enthousiasme et succès, ses recherches de jeunesse sur l'immunité.

Au début de 1896 BÉCLÈRE fut invité par les docteurs BARTHÉLEMY et OUDIN à voir les images formées sur l'écran fluorescent par les rayons de Roentgen. Il décrit sa première impression du nouveau procédé d'exploration par ces mots: »Cette voie me parut le chemin de la Terre promise, je m'y engageai.» En 1896 il se prépara à sa mission radiologique par des études des bases physiques et de la technique instrumentale des rayons de Roentgen, par des examinations radiologiques et par des investigations littéraires.

Le 5 février 1896 il fit, en collaboration avec les Drs. OUDIN et BARTHÉ-

LEMY, sa première communication dans le domaine du roentgendiagnosti[c], »Application de la méthode Roentgen à l'examen d'un anévrisme de l[a] crosse aortique» et dès la même année il organisa, à l'Hôpital Tenon, u[n] enseignement de la radiologie médicale.

Il continua ses recherches dans le domaine du roentgendiagnosti[c], avec une intensité jamais affaiblie, jusqu'en 1910. Cette dernière anné[e], par suite des blessures que lui avaient causé les rayons, il se vit obligé d[e] restreindre son activité radiographique. Depuis lors, il s'est adonn[é] principalement à la roentgenthérapie qui de plus en plus l'enthousias-mait. Après avoir formé de 1900 à 1903 la base scientfique de son activit[é] roentgenthérapique par des études sur la dosimétrie exacte, il commenç[a] en 1904 ses remarquables travaux sur plusieurs branches de la roent-genthérapie. Son œuvre roentgenthérapique n'est ensuite, jusqu'en 1935, interrompue que pendant les années de la grande guerre.

An début des hostilités, en 1914, BÉCLÈRE, dégagé par son âge de toute obligation militaire, proposa ses services. La direction du laboratoire de physiothérapie de l'Hôpital du Val-de-Grâce lui fut confiée. Ensuite, on le chargea d'organiser et de surveiller les laboratoires de radiologie du Gouvernement Militaire de Paris — au nombre de 120 à la fin de la guerre — et de former des médecins radiologistes pour les armées alliées. En l'espace de 4 ans il ne prit que deux permissions de 7 jours.

Après avoir quitté, en 1921, son service à l'Hôpital St. Antoine, BÉ-CLÈRE devint le fervent collaborateur de la Fondation Curie où il conti-nua son enseignement libre de la radiologie médicale jusqu'en 1927. Ses recherches scientifiques et son activité pour la propagation de la radiologie se continuèrent jusqu'à la fin de sa vie longue et glorieuse.

ANTOINE BÉCLÈRE est un des auteurs classiques de la radiologie. Ici ne seront rappelées que ceux de ses ouvrages qui à mon avis méritent d'être qualifiés de fondamentaux.

Le titre de »maître» lui est dû déjà à cause de la manière systématique qui, dès le début, caractérise son activité d'auteur et de professeur. Son premier soin fut d'élucider pour lui-même et ses élèves, par une série d'ouvrages, les éléments physiques et techniques nécessaires pour l'application scientifique de la radiologie. On doit en outre remarquer qu'il a toujours dirigé ses recherches scientifiques, aussi bien dans le do-maine du roentgendiagnostic que dans celui de la radiothérapie, vers les problèmes auxquels l'avaient prédisposé ses études précédentes en matière de pathologie générale et de médecine interne.

BÉCLÈRE se procura lui-même l'instrumentation nécessaire pour ses travaux dans le domaine du roentgendiagnostic clinique, en construisant des *appareils nouveaux pour l'examen fluoroscopique*, appareils ayant servi de modèles à la construction ultérieure d'instruments de ce genre en France et dans une grande partie du reste du monde.

Il introduisit dans la roentgenologie médicale le »*spintermètre*» — détonateur placé en dérivation sur le tube de Roentgen — qui permet de mesurer le pouvoir de pénétration du rayonnement. Il inventa *l'indicateur d'incidence cruciforme* qui permet l'orthodiagraphie rigoureuse pendant la radioscopie.

Ces deux instruments furent, pendant un quart de siècle, indispensables à tout laboratoire de Roentgen. Son »*diaphragme-iris*», qui permet une limitation rapide et précise du champ visuel sur l'écran fluorescent, reste toujours le prototype des limitateurs de rayons primaires employés partout dans la radioscopie.

En 1899, il publia ses *études expérimentales sur la vision dans l'examen radioscopique*, d'une importance fondamentale pour la connaissance du rôle de l'adaptation lumineuse dans la radioscopie. A quel point cet exposé était utile ressort du fait que, bien avant dans le XX^e^ siècle, on pouvait rencontrer des cliniciens éminents qui qualifiaient les observations sur l'écran fluorescent de pures élucubrations, parce que leurs yeux non adaptés ne pouvaient distinguer les phénomènes décrits.

Quoique BÉCLÈRE, surtout dans les premières années, se servit principalement de la radioscopie pour l'examen des organes internes, il fit, déjà de bonne heure, ressortir la supériorité de l'image radiographique sur l'image radioscopique, non seulement en raison de la netteté de l'image photographique, mais aussi à cause des circonstances plus favorables à la vision lors de son interprétation. Déjà en 1899, il désigna les champs d'activité principaux de la radioscopie et de la radiographie tout en montrant leur valeur indispensable pour le diagnostic clinique.

A titre de science, le roentgendiagnostic tend vers un double but. L'un, le plus fondamental, est d'interpréter les radiogrammes, c'est à dire de référer les surfaces et contours des images radiographiques aux parties correspondantes des organes anatomiques. L'autre consiste, une fois que le médecin a appris à posséder la »vue roentgenologique», à mettre la biopsie radiologique au service de la médecine générale, répandant ainsi un nouveau jour sur les phénomènes vitaux à état normal ou pathologique.

BÉCLÈRE fut actif dans ces deux branches du roentgendiagnostic. Mais ses ouvrages principaux visent à interpréter la langue moderne des images roentgeniennes dans les termes classiques de l'anatomie et de la clinique.

Celui qui a vu lui-même combien il était difficile pour la plupart des cliniciens de ce temps-là de saisir que les ombres qu'ils voyaient sur l'écran fluorescent et sur la radiographie correspondaient réellement aux images anatomiques connues des tables d'autopsie et des recherches expérimentales sur les animaux, sait apprécier à sa juste valeur la perspicacité, l'érudition et la largeur de vues de l'homme qui, déjà au tournant du siècle, exposa dans de magistraux ouvrages les bases de

l'interprétation des images roentgeniennes des organes intrathoraciques et abdominaux.

Après avoir publié plusieurs ouvrages spéciaux sur le roentgendiagnostic interne, BÉCLÈRE publia en 1904 un exposé systématique sur »*Les rayons de Roentgen et le diagnostic des maladies internes*» et, en 1913, un rapport magistral sur »*L'exploration radiologique dans les affections chirurgicales de l'estomac et de l'intestin*».

30 ans avant que la volumemétrie radiologique du cœur eût obtenu une place reconnue dans le diagnostic clinique, BÉCLÈRE avait compris son importance et élaboré *une méthode nouvelle de mesurer l'aire du cœur à l'aide des rayons de Roentgen* (1900).

BERGONIÉ et CARRIÈRE avaient décrit en 1899 *le déplacement respiratoire du médiastin au cours des épanchements liquides de la plèvre*, et HOLZKNECHT avait signalé, la même année, ce phénomène dans un cas de sténose bronchique. Tout en confirmant le rôle de la pleurésie et de la sténose bronchique unilatérale, BÉCLÈRE montra, en 1900, *que la sclérose pulmonaire* est en réalité le facteur qui *produit le plus fréquemment le déplacement respiratoire du médiastin*, et il en donna l'explication pathogénique la plus lumineuse. En 1902, il découvrit *les procédés permettant d'explorer radiologiquement les plèvres interlobaires pathologiques* et jeta par là les bases *du roentgendiagnostic des pleurites interlobaires*.

En décembre 1902, en se basant sur des radiographies dont la première fut prise déjà au mois de mai 1901, il décrivit les déformations caractéristiques du crâne et spécialement de la fosse pituitaire lors de l'acromégalie (cas alors vérifié par autopsie). Il attira l'attention sur le fait que la radiographie du crâne deviendrait un des facteurs les plus importants du diagnostic de l'acromégalie fruste.[1] En 1903, il formula à nouveau, dans un article didactique, les lignes principales du »*radiodiagnostic de l'acromégalie*».

Par des ouvrages magistraux, parus de 1901 à 1904, il a prouvé que *les déformations hippocratiques des doigts*, observées dans diverses affections de l'appareil respiratoire, sont liées à une compression des racines ou du tronc de l'artère pulmonaire. Une rétention mécanique, dans le sang veineux des extrémités, des substances toxiques destinées à être éliminées par la voie pulmonaire, est donc la cause probable de »l'hippocratisme digital».

Déjà en 1903 BÉCLÈRE souligna l'importance de l'*examen radiologique au service de l'assurance sur la vie*.

Dès le commencement du siècle, il mit sans cesse les radiologistes en

[1] ARNOLD JOSEFSON avait, le 2 mars 1901 déjà, démontré, à la Société Médicale de Stockholm, une radiographie prise par TOR STENBECK, montrant les traits typiques de la fosse pituitaire acromégalique, communication qu'il publia en suédois au mois de mai 1902 (Hygiea, p. 114).

gde contre *les dangers qu'entraine l'emploi des rayons de Roentgen* et en nintes publications il a préconisé *les moyens de protection* du médecin et des malades contre les nouvelles irradiations.

La *radiothérapie* avec ses branches différentes — la *finsenthérapie*, la *rntgenthérapie* et la *curiethérapie* — ouvrait un champ tout nouveau aux recherches scientifiques. Les recherches empiriques cliniques devançaient ici de beaucoup les recherches expérimentales. BÉCLÈRE, le grand clinicien, a fourni ses plus remarquables apports au domaine de la radiothérapie empirique, où il apparut de nouveau comme savant systématique et comme découvreur.

Ainsi que je l'ai déjà dit, son activité d'auteur dans le domaine de la radiothérapie commença en 1904. D'après ce qu'il fit souvent ressortir lui-même, c'était FRANCIS H. WILLIAMS de BOSTON qui par son ouvrage monumental »*The Roentgen Rays in Medicine and Surgery*» attira son attention sur les possibilités de la radiothérapie. BÉCLÈRE suivit aussi, dans leurs grandes lignes, les principes de WILLIAMS dans l'application de la radiothérapie. Anisi fut-il, d'accord avec WILLIAMS, FREUND et RIEDER, partisan d'un principe de traitement que je voudrais appeler »*principe de traitement biologique*». Il se servit, aussi bien pour les maladies bactériogènes que pour les néoplasmes, de *la méthode de fractionnement et de l'espacement des doses*, tout en prenant en considération dans chaque cas l'effet du traitement sur le procédé de guérison.

D'une grande importance pour le développement de la roentgenthérapie sur une base scientifique furent ses ouvrages de 1902—1906 sur *les mesures exactes et le dosage en roentgenthérapie*. Par une série d'ouvrages sur le même thème, publiés en 1922—1927, il a fortement contribué à *l'unification internationale des mesures de dosage en roentgenthérapie*. Dans un article sur *la radiosensibilité des cellules néoplasiques*, publié dans les *Acta Radiologica* en 1926, il prédit le succès de la méthode de fractionnement dès que l'on disposerait d'une technique de filtrage plus perfectionnée, telle que celle de nos jours.

BÉCLÈRE avait le grand avantage de disposer, dès le début, d'un service hospitalier où l'effet du traitement radiologique pouvait être successivement étudié, jour par jour, au lit du malade. Son service de médecine générale à l'Hôpital St. Antoine peut être appelé, à juste titre, la *première clinique radiothérapique de l'Europe*.

Il a apporté des contributions de grande valeur à la plupart des branches de la roentgenthérapie et donna déjà de bonne heure des exposés critiques sur *l'évolution de la radiothérapie* (1907) et sur *la base scientifique et le domaine de cette science* (1910). Son œuvre scientifique capitale dans la roentgenthérapie se rapporte cependant à *la roentgenthérapie des néoplasmes* et à *l'effet des rayons de Roentgen sur les glandes endocrines*.

Nul plus que lui n'a contribué à la mise en pratique, en France, des résultats scientifiques de la radiothérapie des néoplasmes.

En 1906, il reçut le Prix Daudet de l'Académie de Médecine pour un ouvrage sur *»des résultats obtenus dans le traitement du cancer par l'application des rayons X»* et l'année suivante il donna un exposé général sur *»l'influence des rayons de Roentgen sur les tumeurs malignes»*.

Dans plusieurs travaux, il a vérifié scientifiquement et assemblé d'une manière systématique les résultats de *la roentgenthérapie et de la radio-chirurgie dans les domaines des néoplasmes du sein et des tumeurs de la cavité cranio-rachidienne.*

Pour plusieurs formes de néoplasmes malignes il a été le premier à appliquer, avec succès, la roentgenthérapie. Dans la même année (1904) il réussit *le premier à obtenir une guérison primaire du cancer du larynx et du cancer ainsi que du sarcome maxillaires.* Ce n'est que vingt ans plus tard, environ, que la radiothérapie trouva un emploi reconnu dans le traitement de ces tumeurs.

En 1905, il découvrit *la radiosensibilité extraordinaire des séminomes d'origine testiculaire.* Il a ensuite, jusqu'en 1935, dans une série d'ouvrages devenus classiques, continué ses recherches sur la clinique et la radiothérapie de ces tumeurs. Il prouva que, par la roentgenthérapie, l'on peut empêcher pendant une longue période, même dans des cas présentant d'énormes métastases abdominales, l'apparition de récidives, et qu'après l'ablation d'un néoplasme du testicule, la *roentgenthérapie prophylactique* des récidives est toute indiquée tant sur la région opératoire que sur le domaine des ganglions auxquels aboutissent les vaisseaux lymphatiques de cette région (1916).

En 1934, il vérifia l'observation de FERGUSON au sujet de l'excrétion de prolan A dans les urines des porteurs de séminomes purs et insista sur l'importance de la recherche de cette hormone pour le diagnostic précoce de ces tumeurs et de leurs métastases. En 1935, il montra, en collaboration avec P. GIBERT et H. HINGLAIS, que la présence dans l'urine de *prolan B*, même en minime proportion, isolé ou associé au prolan A, est, en règle générale, l'indice de l'existence d'une tumeur maligne de structure différente du type de séminome pur et d'une très médiocre radiosensibilité. *La présence du prolan B révèle donc l'existence d'une tumeur de pronostic très grave.*

BÉCLÈRE fut ainsi à même non seulement d'ouvrir un nouveau champ d'action à la roentgenthérapie, mais aussi d'élargir la connaissance des indications et du pronostic de la radiothérapie des néoplasmes testiculaires.

Son vieil intérêt pour la physiologie et la pathologie des glandes endocrines l'amena, en 1898, à penser que l'on devrait par l'irradiation d'un adénome du lobe antérieur de l'hypophyse pouvoir faire cesser l'hyper-

sécrétion qui est à la base de l'acromégalie et du gigantisme. Il est *le premier à avoir réussi à guérir par l'irradiation de l'hypophyse un cas d'acromégalie* (présenté à la Société Médicale des Hôpitaux de Paris le 12 février 1909). Il est aussi le créateur d'une technique hardie — irradiation dirigée par plusieurs portes d'entrée à travers le cerveau — dont on se sert encore à l'heure actuelle avec succès dans le monde entier.

Quelques semaines seulement avant que BÉCLÈRE présentât son premier cas d'acromégalie, qu'il traitait depuis décembre 1908, GRAMEGNA avait publié, le 15 janvier 1909, un cas d'acromégalie traité par les rayons de Roentgen. Dans ce cas unique, où GRAMEGNA avait tenté l'irradiation par voie buccale sur la région pituitaire, à l'aide d'un localisateur, il n'avait obtenu qu'une amélioration temporaire. BÉCLÈRE, en poursuivant ses recherches, prouva cependant avec évidence, dans cinq publications parues de 1909 à 1922, la supériorité de sa méthode de traitement pour l'acromégalie. En effet, par la roentgenthérapie de l'adénome hypophysaire d'après la méthode de BÉCLÈRE, l'on obtient dans la plupart des cas d'acromégalie de ce type se trouvant encore en progression, une amélioration rapide non seulement des symptômes causés par une compression locale du nerf optique, mais aussi de symptômes généraux éventuels de compression céphalée, tels que maux de tête et vertiges. Ce traitement arrête les processus hyperplasiques du squelette et des parties molles dus à l'hypersécrétion du lobe antérieur de l'hypophyse, mais ne peut faire disparaître les lésions déjà acquises du squelette. Par contre, on peut graduellement arriver à faire diminuer considérablement l'hyperplasie des parties molles.

Dans des cas avancés d'acromégalie, où l'adénome hypophysaire est déjà en état de dégénération et quand à l'hyperfonctionnement de la glande a succédé une insuffisance fonctionelle amenant le déclin physique et mental, la roentgenthérapie, comme tout autre traitement, n'a aucune chance de succès.

De nombreuses publications d'autres savants ont confirmé les observations de BÉCLÈRE sur la roentgenthérapie de l'acromégalie. Parmi les exploits scientifiques de BÉCLÈRE, l'on peut considérer comme le plus beau celui d'avoir institué et élaboré non seulement le roentgendiagnostic précoce, mais aussi la roentgenthérapie de l'acromégalie.

La radiothérapie des fibro-myomes de l'utérus réalisant la suppression de la fonction ovarienne fut découverte en 1904 par le compatriote ingénieux de BÉCLÈRE, FOVEAU DE COURMELLES. Mais depuis cette époque nul plus que BÉCLÈRE n'a contribué aux recherches scientifiques sur la *roentgenthérapie des fibro-myomes utérins dans ses rapports avec la physiologie et la pathologie ovariennes* et mieux que quiconque il a su faire comprendre aux médecins la valeur pratique de cette méthode.

Après une production de 17 travaux dans ce domaine, parus de 1913

à 1929, il publia sur ses vieux jours, en 1934, un pénétrant ouvrage sur *les bouffées de chaleur de la ménopause spontanée et de la ménopause roentgenienne* où il démontre l'importance de ce symptôme comme indice de la suppression de l'activité ovarienne.

Il s'est également attaqué, dans ses recherches, à la question de la *roentgenthérapie de la maladie de Basedow* (1920). En 1921 il donna, dans le Paris Médical, un intéressant exposé sur les problèmes et les résultats de *la radiothérapie des glandes endocrines.*

S'étant livré pendant une trentaine d'années à l'étude des néoplasmes et de la radiothérapie du cancer, BÉCLÈRE fut forcément amené à s'intéresser vivement au problème relatif à l'*étiologie du cancer.* Par ses observations de la clinique des tumeurs et surtout par l'étude du cancer des radiologistes, il était arrivé à la conviction que le cancer devait être une maladie virulente. Les travaux de PEYTON ROUS sur les ultra-virus causant différents sarcomes des poules et ceux de SHOPE sur un ultra-virus provoquant un papillome chez les lapins sauvages communément appelés »cotton-tails» (queue de coton) l'avaient confirmé dans cette conviction. En 1935 et 1936 il présenta dans la Presse Médicale deux extraits intéressants de ces observations. Jusqu'à sa mort il se consacra, à la Fondation Curie, à des recherches sur l'immunisation contre le virus du papillome de SHOPE. Un des fondateurs de l'immunologie, il s'adonna jusqu'à son dernier jour aux problèmes actuels de cette science.

A ce que je sache, BÉCLÈRE est *le premier à avoir organisé des cours réguliers de radiologie médicale.* Déjà en 1897, il fonda un enseignement de roentgendiagnostic et, à partir de 1898, il donna chaque année, pendant trente ans, des cours libres bi-annuels de radiologie médicale. Au début, ses »conférences de dimanches» sur les rayons de Roentgen se rapportaient aux moyens de production, modes d'emploi et applications au diagnostic médical avec présentation et examen radioscopique des malades. En novembre 1901 la radiothérapie apparut sur le programme. En 1902 le cours libre fut donné pendant huit jours de suite. Chaque jour, à 10 heures du matin, furent enseignées »les notions théoriques et techniques indispensables à la pratique de la radioscopie, de la radiographie et de la radiothérapie», à 11 heures commencèrent des »exercices pratiques de radioscopie, particulièrement appliqués à l'exploration des organes thoraciques» et à 2 heures de l'après-midi des »exercices pratiques de radiographie, simple et stéréoscopique, des diverses régions».

En 1908, quand je visitais, pour la première fois, la clinique de BÉCLÈRE, son enseignement de la radiologie médicale avait atteint un haut degré de développement. Ses cours libres donnés pendant les vacances étaient ouverts à tous les étudiants et docteurs en médecine. En dehors de ces cours de vacances un *enseignement clinique de la radiologie médicale* était donné journellement. Tous les jours, excepté le dimanche, BÉCLÈRE

son chef de clinique donnèrent des leçons sur »l'examen clinique et le traitement des malades justiciables de la radiothérapie ou de la radiumthérapie», et, trois jours par semaine, des conférences sur le roentgendiagnostic clinique avec examens radioscopiques. En outre de l'enseignement régulier, BÉCLÈRE donna presque chaque année des *conférences spéciales sur les applications de la radiologie au diagnostic et à la thérapeutique.* Ainsi donna-t-il déjà en 1906, pendant quatre dimanches de suite, une série de conférences sur *»la radiothérapie du cancer».* Tout cet enseignement théorique fut donné gratuitement. Les cours de vacances ainsi que l'instruction clinique furent complétés par un *enseignement pratique de radiographie et de radioscopie* donné par le chef du laboratoire. A ces exercices pratiques donnait accès un droit de cent francs.

Le cours libre, qui s'étendit au fur et à mesure du développement de la radiologie et finit par comprendre une vingtaine de leçons données pendant un mois, était de nature tout à fait élémentaire. Il fournit de courts exposés de presque toutes les branches de la radiologie médicale: physique, technique et application au diagnostic médical et à la thérapeutique. L'enseignement spécial de la radiologie médicale fut donné dans les cours cliniques de roentgendiagnostic et de radiothérapie.

Bien des années avant que la plupart des pays eussent institué un enseignement régulier, BÉCLÈRE avait ainsi organisé *un enseignement complet de cette nouvelle science.* Ce qui donne à l'enseignement de BÉCLÈRE une place à part, c'est le fait que, dès le début, son enseignement visait, *et à instruire les étudiants en médecine, et à former des radiologistes spécialisés.* Dans les autres pays, l'enseignement pendant les premières dix années ne visait qu'à la formation de spécialistes. Par l'enseignement de la jeunesse, BÉCLÈRE voulut frayer le chemin à la nouvelle science. Dans son pays il a bien rempli les fonctions d'un professeur de Faculté et il a été le *professeur de la radiologie médicale du monde entier.* Durant trente ans il instruisit des milliers d'élèves français et étrangers. Et pourtant la Faculté de Médecine de Paris n'ouvrit jamais ses portes à l'enseignement de BÉCLÈRE. Ceci nous étonna beaucoup, qui considérions comme un grand privilège d'écouter le Maître. Mais ce fait n'indique pas que la Faculté était mal disposée envers BÉCLÈRE personnellement. Les facultés de médecine de France n'ont jusqu'à ce jour qu'exceptionnellement jugé nécessaire de créer des chaires de radiologie médicale.

BÉCLÈRE était un maître dans l'art de l'enseignement, aussi bien par son analyse critique de problèmes scientifiques que dans la présentation aux débutants des notions scientifiques élémentaires. Ses conférences étaient toujours largement illustrées par des tableaux, radiographies etc. et déjà dans les premières années de ses cours, il organisa un petit musée du matériel d'enseignement. Il avait un don tout particulier d'exposer, en des termes précis, les traits essentiels et caractéristiques de ce qu'il

devait démontrer, et de graver dans la mémoire des auditeurs, par des comparaisons spirituelles, les faits démontrés. Il était laconique et pourtant éloquent. On sentait qu'il exposait une conviction acquise grâce à un travail assidu.

BÉCLÈRE fut un pionnier de la radiologie médicale non seulement par ses recherches et son enseignement. Il a aussi contribué à poser une base solide à la radiologie par ses efforts pour réunir les radiologistes à un travail commun pour la consolidation et la propagation de la nouvelle science. Déjà en 1899, alors que la valeur de la roentgenologie n'était comprise que par un nombre insignifiant et que sa mise en pratique était souvent confiée à des physiciens et à des techniciens, il lutta habilement pour que, dans l'intérêt des malades, la pratique de la radiologie soit confiée aux médecins et que tous les hôpitaux soient dotés de laboratoires de radiologie.

BÉCLÈRE était un des fondateurs de la »*Société de Radiologie Médicale de France*» dont il fut le premier président et, plus tard, président d'honneur. Pour procurer à la France une revue d'ensemble de toutes les branches de la radiologie, il fonda en 1914, avec l'aide de neuf jeunes radiologistes prometteurs, le »*Journal de Radiologie*». Dans ce journal, qui dans le monde entier a su acquérir une estime méritée, BÉCLÈRE publia quelques-uns de ses travaux principaux.

BÉCLÈRE suivit avec des yeux attentifs tout ce qui se passait dans le domaine de la radiologie en France et à l'étranger. En 1902, il visita Vienne pour se documenter sur la dosimétrie élaborée par KIENBÖCK et HOLZKNECHT. En 1921, il étudia à la clinique gynécologique d'Erlangen la méthode élaborée par SEITZ et WINTZ pour la radiothérapie profonde du cancer de l'utérus. Ces voyages se traduisirent tous deux par d'instructifs rapports dans les revues françaises. M^elle^ BÉCLÈRE raconte avec quel soin et quelle persévèrance il sut tirer, de la littérature étrangère, toutes connaisances nouvelles utiles à lui-même et à ses élèves. »Ayant quelques éléments d'allemand, aucun d'anglais, il traduisit mot à mot — les écrivant en entier en français — les livres de WILLIAMS et d'HOLZKNECHT. Toujours d'ailleurs il procédera ainsi, parvenant à traduire à livre ouvert ces langues qu'il ne sait parler, accumulant notes, analyses, documents.»

Avec M. JAUGEAS il faisait paraître en 1908 une édition française de »*l'Atlas de radiographie de l'homme normal*» par RUDOLF GRASHEY.

BÉCLÈRE fut un de ceux qui appuyaient le plus la proposition des collègues anglais de réunir les radiologistes de tous les pays à un congrès international à Londres, en 1925. Il fut un *membre très actif et très estimé du »Comité international de radiologie»* auquel il présida de 1928 à 1931. Il fut le *président du 3ème Congrès international de radiologie* siégeant à Paris en 1931. Grâce aux préparatifs assidus et habiles de son président

de ses collaborateurs, parmi lesquels il faut en premier lieu nommer sa secrétaire inlassable M^elle^ ANTOINETTE BÉCLÈRE, ce congrès fut un grand succès et fit le plus grand honneur à la radiologie française. Tous les participants à ce congrès garderont toujours en mémoire le précieux souvenir de la bienveillance, l'amabilité et l'exquise hospitalité avec lesquelles nos hôtes français, en premier lieu le président du congrès, surent nous recevoir.

Le rôle de BÉCLÈRE de »lien agissant» parmi les radiologistes du monde entier, dû à ses rares dons de coordination et à ses éminentes qualités personnelles, ne fit que s'accroître d'année en année. De nombreuses et importantes distinctions honorifiques lui furent conférées par sa patrie comme par l'étranger. Entre autres, il était membre d'honneur de la Société de Radiologie des Pays Septentrionaux dès sa fondation.

ANTOINE BÉCLÈRE réunit en lui toutes les qualités qui forment un grand savant et un bon médecin. Il fut toujours inspiré dans son œuvre par une indomptable passion pour la science, tout en ne perdant jamais de vue que la raison d'être et le but de la science médicale sont d'apporter aide aux malades. Il avait l'imagination constructive, baguette divinatoire nécessaire pour découvrir les sources cachées de la science. Il possédait aussi, à un haut degré, la perspicacité, l'esprit d'analyse critique et la réflexion sereine qui seules permettent de capter ces sources et de les mettre au service de la science. Grand travailleur durant toute sa vie, il réussit, pendant de longues années, grâce à son remarquable esprit d'ordre et à une persévérance admirable, à poursuivre parallèlement plusieurs vastes recherches dans les domaines les plus variés de la médecine, tels que la sérologie, l'endocrinologie, le roentgendiagnostic et la radiothérapie. Ses notes minutieuses sur ses observations personnelles et ses extraits de la littérature forment toute une bibliothèque.

Ses dons de pouvoir embrasser et s'assimiler des connaissances en de vastes domaines firent de lui un maître pionnier de la radiologie médicale. Tout comme les autres maîtres pionniers de cette science, il acquit sa place prédominante non seulement par sa haute et belle intelligence, mais aussi par le courage avec lequel il osa, à une époque où la roentgenologie était encore considérée comme une activité accessoire assez douteuse chez un médecin, sacrifier une situation sûre et considérée dans une des disciplines reconnues de la médecine, pour s'adonner entièrement au service précaire et aléatoire de la nouvelle science. Parmi ces maîtres pionniers ANTOINE BÉCLÈRE occupe cependant une place de tout premier plan, et parce qu'il se mit à l'œuvre mieux préparé que tout autre du fait de ses connaissances profondes de la pathologie générale, et parce que, en qualité de chercheur et de professeur, il sut garder un rôle prépondérant dans l'histoire de la radiologie jusqu'en ses pages les plus récentes. Il fut un grand professeur non seulement en écrit comme en paroles, mais aussi

par la sagesse et la bonté qui le caractérisaient et par l'organisation modèle d'enseignement et d'instruction qu'il sut créer et diffuser.

Sa passion vivace et desintéressée pour la science médicale et son culte de la mission du professeur l'ont conduit à sa haute position dans la médecine. Un ardent désir de prodiguer son savoir et son expérience rayonnait de toute sa personne et se réflétait dans ses yeux d'un regard bon et vif. Il avait aussi le don particulier d'observer et de s'intéresser à ses élèves. A ceux chez qui il découvrait un amour véridique de la science radiante, il accordait une amitié paternelle qui se prolongeait la vie durant.

ANTOINE BÉCLÈRE ayant beaucoup à donner, répandit aussi à pleines mains. A une époque pleine d'inquiétude et de rivalités, il créa un centre d'équilibre et d'entente. Il fut un de ces êtres privilégiés qui savent enrichir la vie pour tout leur entourage. Ambassadeur serein de la lumière, il nous a donné l'espoir qu'un jour viendra où la raison et la charité deviendront les étoiles conductrices de l'humanité.

Gösta Forssell.

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FROM VANFÖREANSTALTEN (PROF. H. WALDENSTRÖM) STOCKHOLM AND NYNÄS KURANSTALT (DOCTOR F. SUNDELIN), NYNÄSHAMN

THE SEDIMENTATION OF OIL BY MYELOGRAPHY AND ITS DIAGNOSTIC SIGNIFICANCE[1]

by

Folke Knutsson

In carrying out myelography with oil (lipiodol) it is very important to consider the physical conditions of the oil in the cerebrospinal fluid. The position and movement of the oil in the dural cavity is determined by its specific gravity, which causes the oil to sink to the bottom of the canal filled with fluid. Because of its oily consistency the whole quantity of oil forms a big coherent drop with rounded contours or it is divided into several smaller drops of the same nature. Further its intense contrast-density is of importance because a very thin layer is able to give maximal absorption of rays and thus cause the same total defect on the film as a thicker layer would do. In consequence of this intense contrast-density no variations in the intensity of the contrast shadow are caused, and thus the defect will always be maximal. These physical qualities of the oil are to a certain extent a disadvantage, as they do not allow of the contrastfluid forming a true cast of the content in the dural cavity. But under certain conditions these qualities are an advantage, for instance by proving disc-protrusion and other similar low changes of »relief» in the dural cavity, to which I shall revert in the following.

My intention is to illustrate the significance of these physical conditions in connection with myelography with oil by showing a certain number of myelographies carried out in cases of disc-protrusion to the dural cavity. The contrast medium has been 40 % lipiodol of which 3—5 ccs. has been injected.

I have made a model of the lumbar canal by putting a halved wooden cylinder into a glass tube. On the plane surface of the cylinder a little semicircular wooden ball has been placed, which is supposed to represent

[1] Paper read before the Northern Association for Medical Radiology, at the meeting in Oslo, June 1939; submitted for publication Oct. 2, 1939.

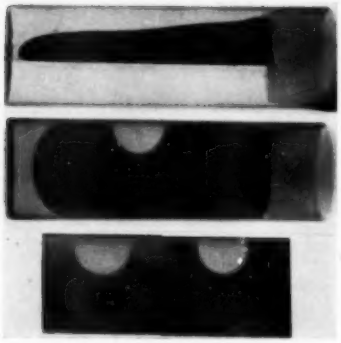

Fig. 1. On the two upper pictures the model is photographed with horizontal and vertical ray-direction. With vertical ray-direction the semi-circular prominence appears, but with horizontal ray-direction it does not appear as a defect or even as a thinning of the contrast-shadow. Observe the evenly rounded oil-pole which is due to the oilyconsistency of the medium, and thus does not imply any change in the relief of the bottom. On the lower picture taken with vertical ray-direction one can notice the change in the size of the defect in proportion as the thickness of the oil-layer changes.

the protrusion from the plane anterior wall of the dural canal. This glass cylinder I have filled with water and lipiodol in suitable proportions and closed the opening with a rubber plug. I have then been able to study the movements of the oil in the water, and by means of roentgenrays discover the conditions for the genesis of a defect in the contrast shadow due to the protrusion (fig. 1).

The importance of the oily consistency of the contrast medium.

These experiments (fig. 1) prove that the oil tends to move as a coherent drop, which on account of surface tension has evenly rounded convex contours. Thus such a convex pole on the contrast shadow does not imply a corresponding obstacle to the movement of the oil, but is due simply to the physical qualities of the latter in water. If, on the contrary, there is an obstacle to the passage of the oil, for instance a low thresholdlike eminence, the oil will be stopped until it has increased in thickness to the point where it will well forth as a single big drop or as several smaller drops. In such a case the height of the pole of the contrast shadow at the obstacle is no true measure of the obstacle itself. A relatively low obstacle may correspond to a high pole of oil. It is necessary to understand this, otherwise one may be tempted to over-estimate the degree of the obstacle (protrusion) (fig. 2, 3, 5a).

On account of surface tension there will be a characteristic meniscus formation where the oil contacts with the walls of the dural canal. The upper surface of the oil-layer is not plane, at the wall it shows an upward convex rounding, making the contact between oilsurface and wall begin a little below the upper horizontal level of the oil-layer. In a case of low prominence the filling defect will, on account of this phenomenon, be somewhat bigger than it would have been had a non-oily contrast-medium been used.

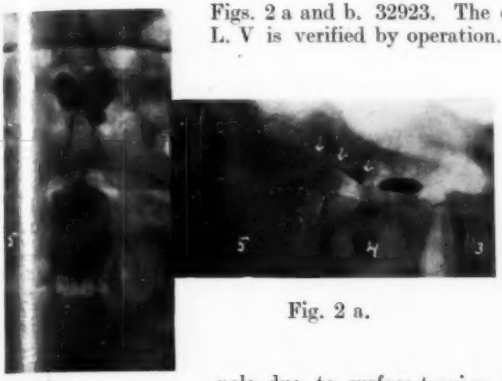

Fig. 2 a.

Figs. 2 a and b. 32923. The disc-protrusion between L. IV and L. V is verified by operation. On the frontal film taken with vertical ray-direction there is a wide break in the contrast layer level with the intervertebral space and the lower part of corpus vertebrae L. IV.

On the lateral film taken with horizontal ray-direction the protrusion appears, which in fig. 2 a is marked by a streak of contrast-medium. It is clear that the protrusion is not high enough to correspond to the distally situated oil-mass. This oil-mass has a rounded cranial pole due to surface-tension and only when it has gained thickness enough does the oil well forth, passing the obstacle. Thus the defect is not due to a complete obliteration of the lumen. The degree of the prominence must not be over-estimated. During the operation it was found to be 0.5 cm.

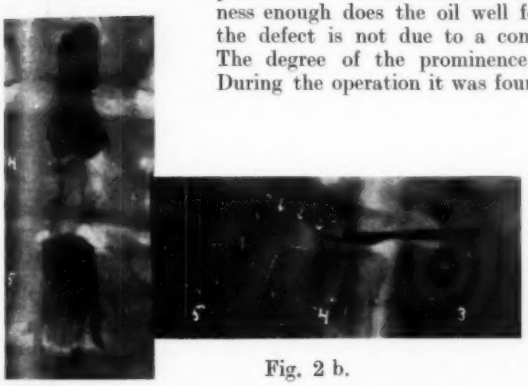

Fig. 2 b.

The significance of the sedimentation.

It is obvious that the sinking of the oil to the bottom of the fluid-filled dural cavity necessitates an appropriate position of the patient in order to make possible a proper examination of the part of the dural canal in question. One must never forget that the oil does not altogether fill the canal but only forms the bottom-layer of its fluid content.

One may easily think that by using horizontal ray-direction the lower surface of the oil-layer could be reproduced and thus one would get an idea of the bottom-relief. If there is a transverse eminence of the bottom along its whole width, then there will be a corresponding defect of the bottom-contour of the oil on the film (fig. 2, 3). If, on the contrary, this eminence only comprises part of the width of the oil-layer, it will not develop a corresponding defect of the lower contour of the oil-shadow. The remaining width of the oil-layer, which is to be found next to the eminence, will form the lower contour. Thus it is only possible to judge profile pictures in combination with films taken in the same position with

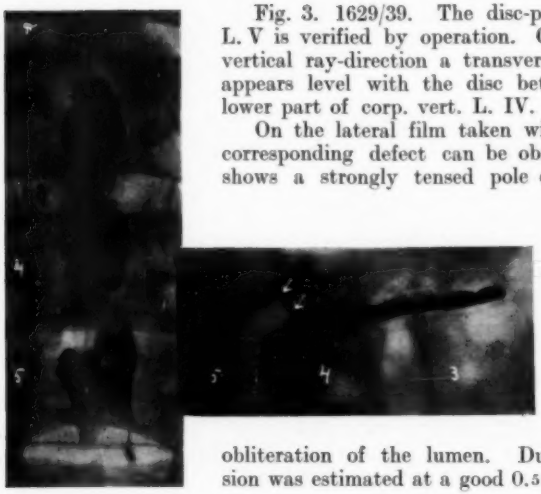

Fig. 3. 1629/39. The disc-protrusion between L. IV and L. V is verified by operation. On the frontal film taken with vertical ray-direction a transverse break of the contrast-layer appears level with the disc between L. IV and L. V and the lower part of corp. vert. L. IV.

On the lateral film taken with horizontal ray-direction the corresponding defect can be observed. The distal oil-portion shows a strongly tensed pole due to surface tension. From here the oil drops down on the other side of the protrusion like a waterfall. Thus the pole-shaped contour is only due to the surface tension of the oil and the protrusion only reaches the declivous contour of this pole. One must not over-estimate the size of the protrusion and still less imagine complete obliteration of the lumen. During the operation the protrusion was estimated at a good 0.5 cm.

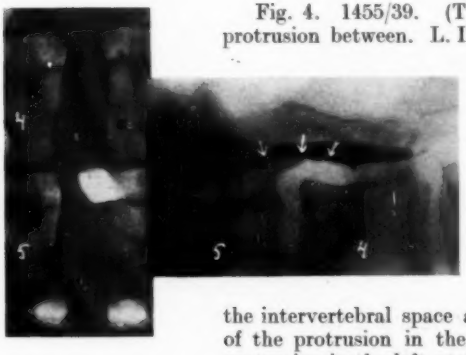

Fig. 4. 1455/39. (The same case as fig. 10.) The disc-protrusion between. L. IV and L. V is verified by operation. On the frontal film taken with vertical ray-direction an even margin defect appears in the left part level with the disc between L. IV and L. V, indicating protrusion. On the lateral film taken with horizontal ray-direction a flat impression of the contrast-layer appears. This impression corresponds to the position of the streak of contrast-medium which passes the intervertebral space and thus forms a measure of the degree of the protrusion in the right part. Thus the degree of the protrusion in the left part can not be estimated (compare fig. 6).

vertical ray. It is the intense contrast-density of the oil that makes it impossible to notice a partial defect. The total ray-absorption of the thin surrounding oil-layer altogether hides this defect (figs. 4, 6).

It is of fundamental importance to understand how the filling defect of the oil-layer will arise where vertical ray-direction is used. In roentgenology we have been accustomed to regard the filling defect in a fluid-filled cavity as a true measure of the shape and volume of the object. Thus the defect directly corresponds to the displacement. So, for example, a gall-stone in a gall-bladder filled with contrast-medium will give a filling

Fig. 5. 2021/39. The disc-protrusion between L. V and S. I is verified by operation. Similar protrusion also between L. IV and L. V.

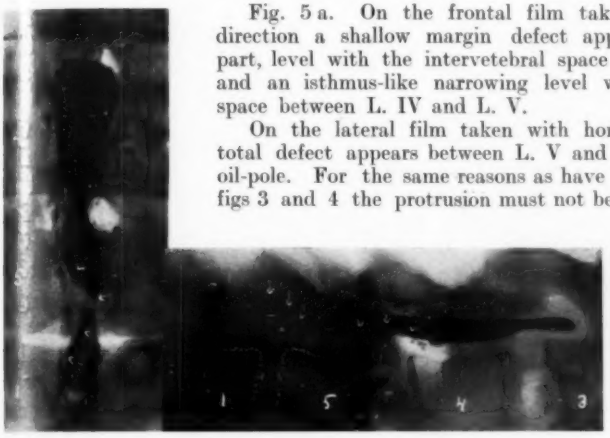

Fig. 5 a. On the frontal film taken with vertical ray-direction a shallow margin defect appears within the right part, level with the intervetebral space between L. V and S. I, and an isthmus-like narrowing level with the intervertebral space between L. IV and L. V.

On the lateral film taken with horizontal ray-direction a total defect appears between L. V and S. I with a high lower oil-pole. For the same reasons as have been given concerning figs 3 and 4 the protrusion must not be over-estimated. There is also a protrusion between L. IV and L. V. Apparently we have here to deal with bilateral prominences and the film only shows the degree of the protrusion in the centre part where it is lowest.

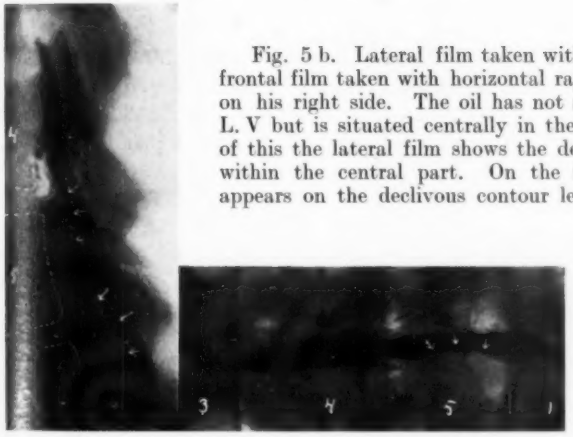

Fig. 5 b. Lateral film taken with vertical ray-direction and frontal film taken with horizontal ray-direction and the patient on his right side. The oil has not sedimented within L. IV—L. V but is situated centrally in the dural cavity. On account of this the lateral film shows the degree of the disc-protrusion within the central part. On the frontal film the protrusion appears on the declivous contour level with L. V.

defect which exactly corresponds to its true shape and size. That is why the gallstone appears through the contrast-medium.

But should the gallbladder be filled with lipiodol it would not be possible to see the stone as a defect if it had not direct contact with the walls so that it was not altogether surrounded by oil. The same comparison can be made by examination of polyps in the alimentary canal where they appear as defects because of the possibility to expose through the contrast-medium.

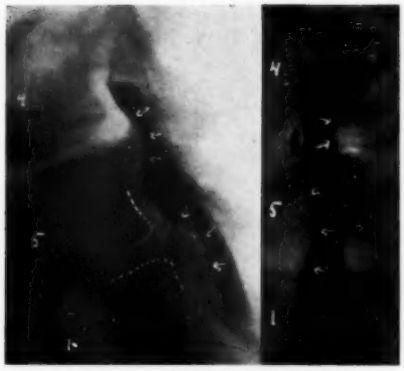

Fig. 5 c. In the standing position the dural cavity is totally filled with contrast-medium to a cylindrical contrast-pillar.

The defect to be found in the oil-layer by myelography where there is a prominence of the anterior wall is theoretically of quite another kind. I do not think that in this connection the mecanism has been made clear.

We imagine a patient in the prone position with a semicircular protrusion of the anterior wall of the dura because of disc-protrusion and the lipiodol is located at the protrusion. We take the film with vertical ray-direction. Then we get a round defect in the contrast-shadow just as if we had placed a vertical cylinder in the oil-layer. The defect is total and the surrounding contrast-shadow is maximal. There is no successive transition from thin to dense contrast-shadow. *The filling-defect is exclusively due to that part of the prominence that rises above the fluid-level. The contour of the defect is caused by the borderline between the oil-surface and the prominence* (*fig. 7*). By way of comparison we may take a shoal in a lake. We can only see that part of the shoal that is above the surface of the water. Its size will vary according to the level of the water, and it would be wrong to judge the total size and shape of the shoal by the part that rises above the water. This is also the case with the conditions disclosed by myelography. The defect in the oil-shadow is due to the part of the prominance (the shoal) that rises above the oil-surface. The contour is due to the defect of the upper surface of the oil-layer (the shore) and its width and course is due to the thickness of the oil-layer. Thus from the size of the filling-defect one is not entitled to draw any conclusions as to the height of the protrusion. The only thing one can be certain about is that

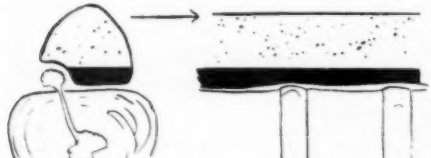

Fig. 6. Diagram showing the genesis of the margin defect in case of disc-protrusion on the frontal film with vertical ray-direction and its absence on the lateral film with horizontal ray-direction.

Fig. 7. The prominence rises as a shoal over the oil-level. By photographing with vertical ray-direction a round defect appears in the contrast-shadow, due to the defect in the upper surface of the oil. The contour of the defect is caused by the defect in the surface layer. It is just like a shoal which rises above the surface of the water. The defect is caused by that part of the »rock» above the »water» and its contour corresponds to the »shore». One can not draw any conclusions as to the size of the »submerged rock».

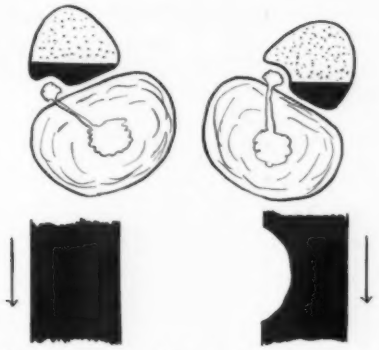

Fig. 8. The conditions for the margin defect in case of disc-protrusion and examination in half-inclined positions in the prone position. It is the movement of the oil-layer and its varying thickness that causes the variations in the size of the defect and its total disappearance.

there is a protrusion of the bottom, the upper part of which is rounded, oval etc. Thus the size of the defect is to a great extent due to the thickness of the oil-layer.

In half-inclined positions of the patient in the prone position the oil sinks to one of the two sides, and the contrast layer varies in thickness and thus the size of the filling defect also varies (figs. 8, 9). In these changes of position the variation of the size of the defect is not only due to the anterior or anterio-lateral position of the protrusion as has generally been understood (LOVE, CAMP) but is due to changes in the thickness of the oil-layer brought about by the changes of position. The extent of this defect is, naturally, influenced by the size of the protrusion as LOVE and CAMP rightly point out, but the defect is to just as great an extent due to the thickness of the oil-layer. On the other hand BRUUN has clearly seen how the defect arises. He writes: »When judging the size of the disc-protrusion one must take into consideration the thickness of the lipiodol-layer. Where the layer of lipiodol is thin, quite small or low protrusions may cause great margin defects.» In this respect the oily consistency of the contrast medium is a real advantage. One can clearly and distinctly see the low protrusion as a total filling defect. By using another contrast-medium, which would cover the whole protrusion with a layer, one would only be able to see the prominence as a

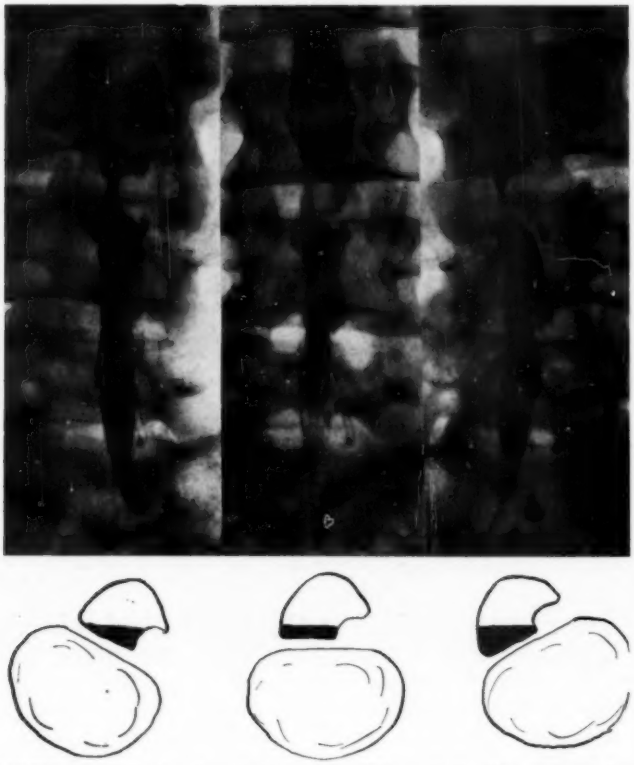

Fig. 9. 4042/37. Disc-protrusion between L. III and L. IV verified by operation. The margin defect varies in size on changing from frontal position to half-oblique position in the prone position. (Compare fig. 8.)

diffuse thinning, which would perhaps be difficult to observe. For the same reason myelography with air is greatly inferior myelography with oil.

Such a distinct round filling defect has the same appearance as the defect caused by intradural rounded tumour, but complementary photography with horizontal ray-direction enables one to distinguish between them. The intradural tumour still causes the same distinct round filling-defect, while on the other hand the extra-dural protrusion appears as a low prominence of the bottom layer of the lipiodol (REISER and others).

To prevent a misinterpretation of the roentgen on account of the sedimentation of the oil one ought to photograph the patient in the same position with both horizontal and vertical ray-direction.

On an ordinary lateral-film taken with vertical ray-direction as in fig. 10 one must not be misled into drawing conclusions concerning the anterior wall of the dural canal, for the oil has sedimented within L. I—L. IV down into the declivous part and thus does not show any cast of the anterior wall. Within L. IV—L. V on the contrary the oil is to be found in the central part of the canal on account of discprotrusion with obliteration of the lumen and consequent hindrance to the sedimentation. Within this part the oil forms a cast of the anterior wall and the discprotrusion appears distinctly as a defect in the contrast-shadow. The frontal film is taken with left side down and on the film the defect appears on the lower contour of the contrast-shadow level

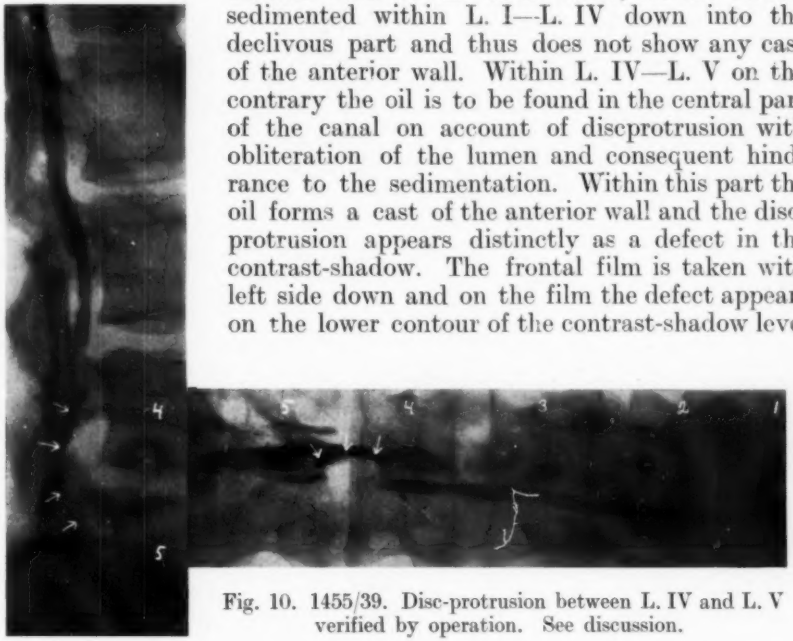

Fig. 10. 1455/39. Disc-protrusion between L. IV and L. V verified by operation. See discussion.

with L. IV—L. V due to the disc-protrusion being localised to the left side (same case as fig. 4).

In fig. 5 b the contrast-medium within S. I—L. IV is to be seen in the central part and only at L. III does the sedimentation start. Thus the lateral film taken with vertical ray-direction allows us to judge the anterior wall of the dural cavity only within S. I—L. IV. Here the examination entitles us to diagnose disc-protrusion between L. IV and L. V and between L. V and S. I. The frontal film is taken with right side down. On the lower contour level with the inter-vertebral space L. V—S. I.

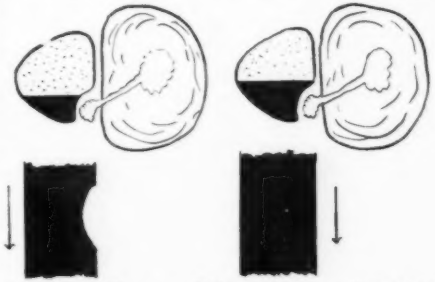

Fig. 11. The conditions for manifestation of disc-protrusion as a defect in lateral film with vertical ray-direction.

there appears a flat impression due to the localization of the protrusion to the right side as appears in fig. 5 a. In fig. 5 c the same case is examined in the standing position. Here the dural canal is altogether filled with contrast-medium and the diagnosis is simple. In fig. 11 the conditions under which a disc-protrusion appears on the lateral film with vertical ray-direction are schematically reproduced. Here the appearance of the defect is due to the thickness of the contrast layer.

The significance of the intense contrast-density of the oil.

As has been pointed out above, the contrast-density of the lipiodol is so intense that even very thin layers give a total absorption of the rays and on the roentgen film there is no gradation to be found between thin and thick layers of contrast-medium. Thus there do not exist any thinnings of the contrast-shadow but only total defects. The consequence of this is that low protrusions, that do not rise through the whole oil-layer but only form »submerged rocks», as it were, do not cause any change of the contrast-shadow on films taken with vertical ray-direction.

With horizontal ray-direction a protrusion that only runs along part of the whole width of the oil-layer, may be altogether hidden by the remaining oil-layer which is to be found at the side of the protrusion and on a planer bottom layer. Then the protrusion does not cause any thinning of the contrast-shadow.

SUMMARY

In carrying out myelography with oil (lipiodol) it is very important to consider the physical conditions of the oil in the cerebrospinal fluid (specific gravity, oily consistency, intense contrastdensity). It is necessary to photograph the patient in the same position with both horizontal and vertical ray-direction in order to prevent a misinterpretation of the roentgen on account of these characters of the oil.

ZUSAMMENFASSUNG

Bei Myelographie mit Öl (Lipiodol) ist es wichtig, dass man die physikalischen Eigenschaften berücksichtigt, die das Öl in der Zerebrospinalflüssigkeit zeigt (spezifisches Gewicht, Ölkonsistenz, intensive Kontrastdichtigkeit). Um die Fehlerquellen beherrschen zu können, welche durch die physikalischen Eigenschaften des Öls bei der Röntgenuntersuchung veranlasst werden, soll man den Patienten in derselben Lage sowohl mit vertikaler als auch mit horizontaler Strahlenrichtung photographieren.

RÉSUMÉ

Dans la myélographie à l'huile (lipiodol) il est très important de tenir compte des caractères physiques de l'huile dans le liquide cérébrospinal (poids spécifique, consistance,

'ensité contrastante intense). Pour se rendre maître des fautes d'interprétation auxquelles 's caractères physiques de l'huile peuvent donner naissance à la radiographie, on doit hotographier le malade dans la même position avec des rayons verticaux et horisontaux.

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FROM THE ROENTGEN DEPARTMENT OF ÅSÖ HOSPITAL, STOCKHOLM

ARTHROGRAPHY AND ROENTGENOGRAPHY IN RUPTURES OF THE TENDONS OF THE SHOULDER JOINT[1]

by

Knut Lindblom

Ruptures of the tendons of the shoulder joint have long been known to anatomists thanks to a work by SMITH in 1934. Publications by CODMAN, AKERSON, KEYES, MEYER, and SKINNER contributed to the knowledge of the pathologic anatomy. In the fields of clinical and roentgenologic symptomology and of therapy CODMAN was the pioneer. His work was continued by BRICKNER, WILSON, FOWLER, ROGERS, HAGGART and ALLEN, FERGUSON, DAVIS and SULLIVAN, MAYER, ELLIS, OUTLAND and SHEPHERD, and GRAY. Among these MEYER was mainly concerned with ruptures of the biceps tendon and partial avulsions of the subscapularis tendon from the lesser tubercle. The other authors were chiefly interested in the so-called supraspinatus ruptures.

The roentgenographic symptoms described by CODMAN as belonging to the disease in question referred to direct roentgenography and consisted of osteophytic deposits, bone atrophy and eroded areas on the greater tubercle. These applied to the inveterate cases, no changes being seen in the recent ones.

The use of arthrography or bursography in the diagnosis of injuries to the tendons of the shoulder was suggested by CODMAN, but he himself was unable to reach any demonstrable results. OBERHOLZER made arthrograms of the shoulder in order to study the lesions of the inferior recess of the humeroscapular joint caused by dislocation. In a recently published monograph on arthrography, in which 3 such cases and 2 normal ones were reported upon, OBERHOLZER made no mention of ruptures in spite of the fact that the figures show ruptures of the tendon aponeurosis in one of the »normal» and two of the »pathologic» cases.

[1] Submitted for publication Oct. 6, 1939.

The rupture was designated by OBERHOLZER as a superior recess of the joint cavity.

Since April 1938 a method of arthrographic diagnosis of the tendon ruptures in question has been used by LINDBLOM and PALMER, and the subject discussed in three clinical and roentgenologic publications. The object of the present paper is to give a more complete report on the roentgenology of the disorder.

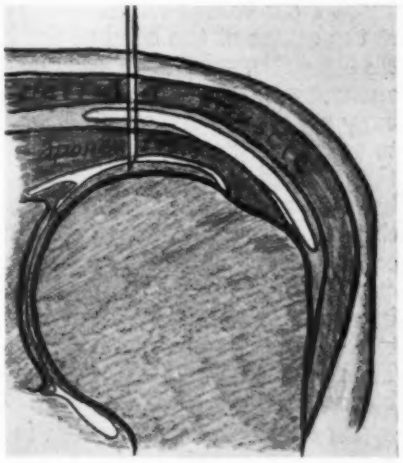


Fig. 1. Sketch illustrating the route of the needle into the joint.

Instead of using the terms supraspinatus, infraspinatus and subscapularis ruptures, the writer refers to ruptures in the respective parts of the tendon aponeurosis of the shoulder joint, as was done in the papers by LINDBLOM and PALMER. By the tendon aponeurosis, on the other hand, is meant the continuous mass of fibrous tissue formed by the tendons of the short rotators and abductors of the shoulder and the coracohumeral ligament.

Material

The material consists of:

54 clinical cases of arthrographically diagnosed ruptures in the tendon aponeurosis of the shoulder joint or in the long biceps tendon. Operation[1] was performed in 20 cases.

50 clinical cases, examined arthrographically and found to have no rupture of the tendon aponeurosis or of the long biceps tendon. Operation was performed in 2 cases.

3 clinical cases examined bursographically, in which no rupture could be demonstrated.

38 anatomical specimens, dissected, roentgenographed, and in the majority of cases examined arthrographically and histologically.

Arthrographical technique

The patient lies on his back with his arm adducted and relaxed. A hypodermic needle 1 mm in diameter and with a short point inserted

[1] Surgeon: Dr PALMER, Sabbatsbergs Hospital, Stockholm.

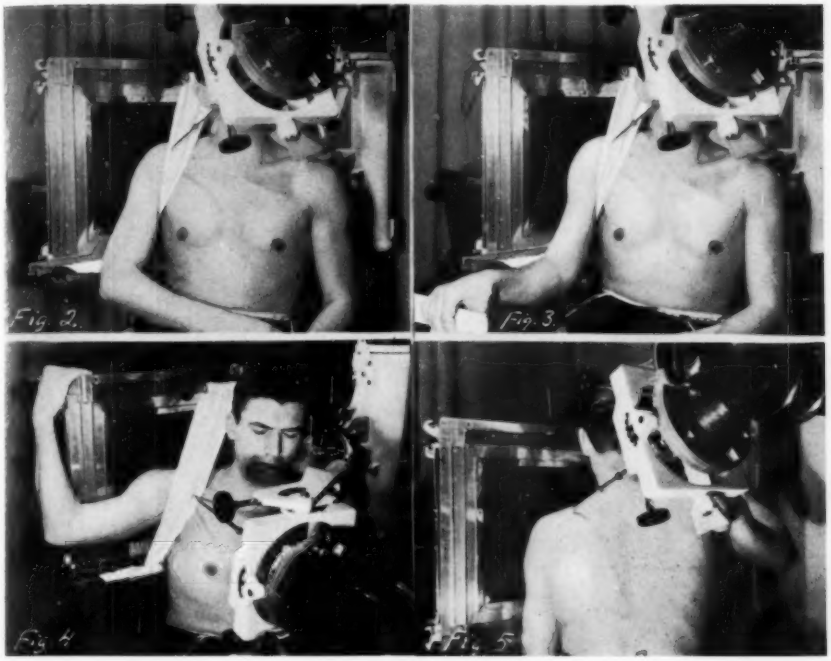
about 1 cm ventro-lateral to the acromioclavicular joint in the direction of the centre of the head of the humerus. The point of the needle pierces the cutis, subcutis, fascia, deltoid muscle, subacromial bursa, tendon aponeurosis, joint space and articular cartilage (Fig. 1). Then it meets bony resistance, the surface of the head. The patient is now instructed to relax completely, and attempts to inject novocain are made. When the point of the needle is in the cartilage, the injection often is balked, but the resistance ceases, if, under continued pressure on the piston, the needle is withdrawn about a millimetre, i. e. to the joint space. The contrast medium, 6 cc. of 35 % perabrodil mixed with 1 cc. of novocain, is now injected, with the patient still relaxed. The needle is removed immediately, and a number of passive movements of the arm are made, in order to spread the contrast medium throughout the joint. The roentgenograms are taken without delay, since the contrast medium becomes rapidly absorbed.

As a rule the intervention is associated with moderate pain as the tendon aponeurosis is pierced. The patient is prepared for this, so that no involuntary movement may break the needle or result in unnecessary injury to the tendon. The injection of the contrast medium, on the other hand, causes no pain, unless the fluid is injected in the tissue of the tendon. Should pain be felt, the injection is continued with the aid of fluoroscopy. If the patient relaxes, the contrast medium flows so easily into the joint that fluoroscopy is unnecessary,

In exceptional cases fluid was withdrawn at puncture; otherwise the aponeurosis was too closely pressed to the head, even when the patient relaxed his muscles. The cases in which fluid was found were of large ruptures. In subacromial bursitis, fluid could be obtained from the bursa during the passage through it. In one case of recent rupture, the synovia was tinged with blood, while in the others it was clear and contained but a few cells of the mononuclear type.

Roentgen technique

The roentgenologic examination was done on LYSHOLM's skull table, which rendered ut easier to obtain exact projection when using a secondary diaphragm. The patient was sitting up with the posterior or antero-lateral part of the shoulder resting against the table. Three positions were used in order to provide tangential views of the insertions of the infraspinatus, supraspinatus and subscapularis tendons or, more exactly, the corresponding parts of the tendon aponeurosis. In exceptional cases a fourth position was added, giving a picture of the joint seen from the glenoid cavity.



Figs. 2—5. Photographs of positions I—IV.

I. (Fig. 2). The scapular region against the table, the arm adducted and not rotated as in the position of rest. Direction of rays: 20 to 26 degrees from above.

II. (Fig. 3). As above but with the arm in about 70 degrees outward rotation.[1]

III. (Fig. 4). The interscapular region against the table and the arm abducted to 90 degrees, if possible, and rotated outwards to 90 degrees. Direction of rays: 10 to 16 degrees from below.[1]

IV. (Fig. 5). The antero-lateral surface of the shoulder against the table, and the arm adducted and rotated outwards. Direction of rays: 20 to 26 degrees from above.

[1] In cases of immobility of the shoulder this position may be obtained by turning the patient or changing the direction of the rays.

The normal arthrogram

In normal cases the injection resulted in the contrast medium filling the joint cavity, the tendon sheath of the biceps, and the subscapularis recess. It is of importance that the subacromial, subdeltoid and subcoracoid bursa never, under normal conditions, became filled from the joint. The appearance of the arthrogram in the various positions is described below.

Position I. (Fig. 6). A thin layer of contrast medium covers the articular surface of the head from the superior part of the anatomical neck down to the inferior recess. The inferior recess is continued as a recess covering the posterior part of the anatomical neck. The subscapularis recess appears as a deposit of contrast fluid under the base of the coracoid process. A band-shaped defect in the filling, corresponding to the biceps tendon, runs through the joint cavity from the superior edge of the glenoid cavity latero-inferiorly. More peripherally the biceps tendon is accompanied a few centimetres from the joint cavity by lines of contrast medium corresponding to the sheath of the tendon.

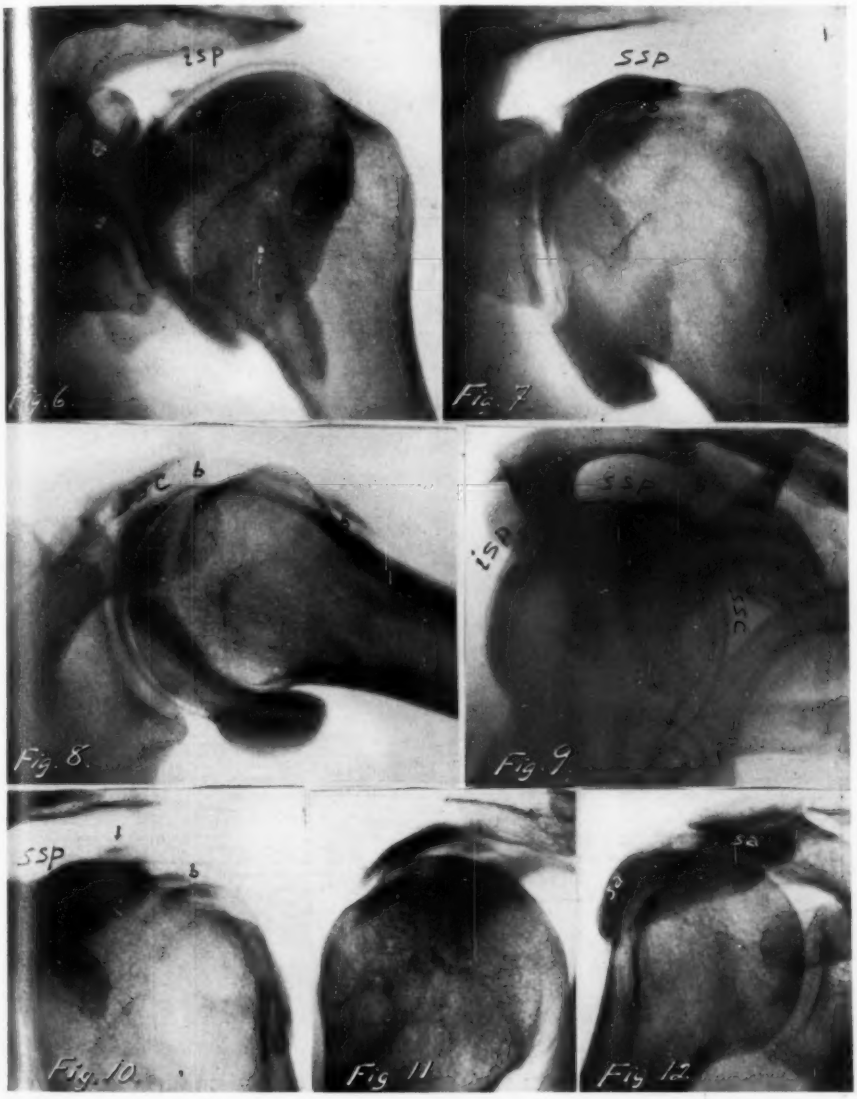
Position II. (Fig. 7). A thin layer of contrast medium covers the articular surface of the head from the superior part of the anatomical neck inside the supraspinatus insertion of the greater tubercle to the inferior recess. The subscapularis recess is compressed by the subscapularis muscle and appears as a thin layer covering the lower surface of the coracoid process. The biceps tendon is represented by a filling defect, starting from the upper edge of the glenoid cavity and continuing first laterally to the upper part of the anatomical neck and then caudally through the tendon sheath.

Position III. (Fig. 8). In this projection also there is a layer covering the articular surface of the head from the anatomical neck at the lesser tubercle to the inferior-posterior recess. The biceps tendon is projected beyond the layer of contrast covering the head, and the tendon sheath lies on the outer aspect of the surgical neck.

Position IV. (Fig. 9). A thin layer of contrast surrounds the head and intra-articular part of the biceps tendon, and an offshoot corresponding to the subscapularis recess runs under the base of the coracoid process.

Technical errors

If the contrast medium is injected into the tendon tissue, a diffusely outlined deposit is obtained which, if enough contrast medium is injected, spreads into large parts of the aponeurosis and takes on its form (Fig. 11).



Figs. 6—9. Normal arthrograms obtained in positions I—IV. Abbreviations: isp, ssp, and ssc = infraspinatus, supraspinatus, and subscapularis portions of the tendon aponeurosis; b= long biceps tendon.

Fig. 10. Normal arthrogram obtained in position II with too great outward rotation of the arm; see text. Arrow marks a leakage through the canal of insertion.

Fig. 11. Roentgenogram of normal case with contrast medium injected in the tendon aponeurosis.

Fig. 12. Normal bursogram of subacromial bursa (sa).

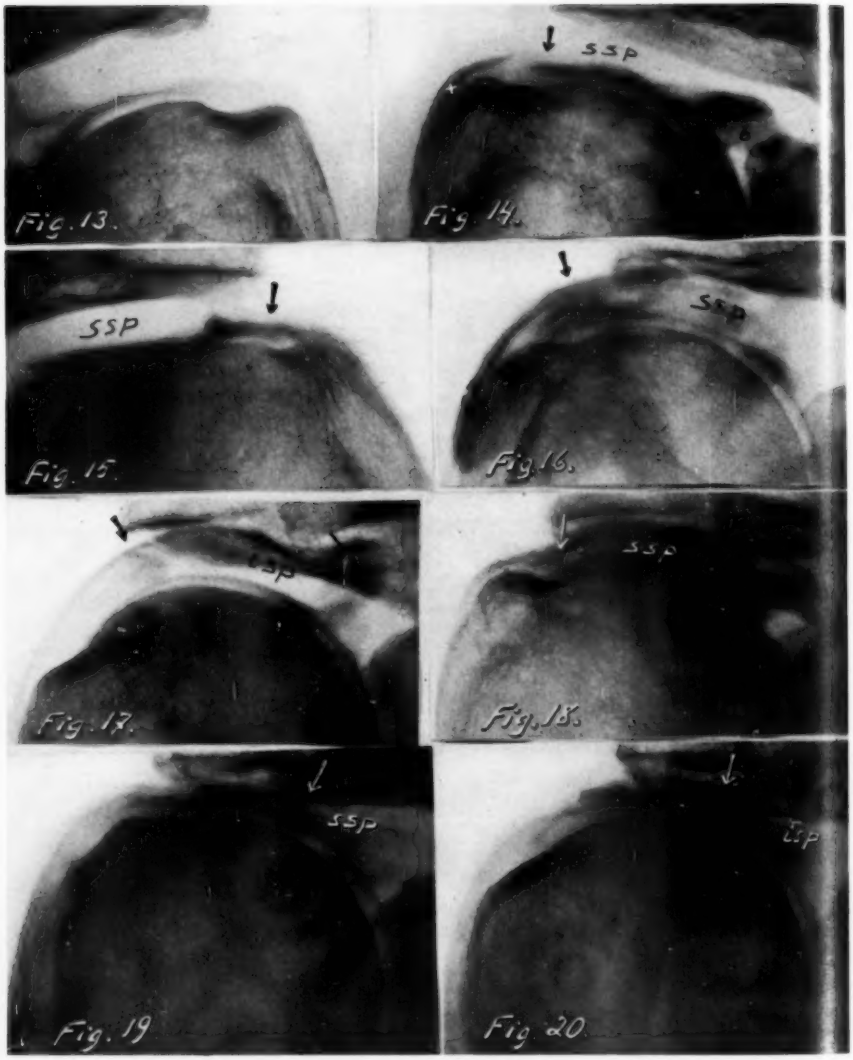


Fig. 13. Normal arthrogram of right shoulder for comparison with arthrogram of left shoulder in same case shown in Fig. 14.

Figs. 14—20. Arthrogram in ruptures of the supraspinatus and infraspinatus portions of the tendon aponeurosis (See text). Arrows mark ruptures. x in Fig. 14 marks a callous formation. Abbreviations under Figs. 6—12.

The contrast medium may leak from the joint cavity through the passage caused by the needle into the tendon tissue, forming a small deposit above the joint cavity (Fig. 10). If the above-mentioned instructions regarding the site and direction of the injection are followed, this deposit of contrast medium will be situated a long distance medial to the greater tubercle and cannot be confused with the contrast filling of the rupture cavity in the aponeurosis. When in doubt as to whether the contrast-filled space in the tendon tissue is artificial or preformed, another examination should be made.

If the contrast medium is injected in the subacromial bursa, a bursogram is obtained (Fig. 12).

Too great outward rotation of the arm in Position II causes the biceps tendon to be projected beyond the head (Fig. 10) and the picture to resemble the one in Position III.

Arthrograms in ruptures of the supraspinatus and infraspinatus portions of the tendon aponeurosis

(51 clinical cases, 20 of which were operated upon, and 9 autopsy cases.)

In general ruptures of the tendon aponeurosis of the shoulder were situated near the greater or the lesser tubercle and appeared to have begun in the part of the tendon tissue lying nearest the head. Thus, instead of finishing at the anatomical neck, the contrast filling of the joint continued into the tendinous tissue and more or less covered the tendinous insertion. The anatomical specimens showed that the joint capsule is intimately connected with the tendon aponeurosis, particularly its supraspinatus portion, and that the same is true of the floor of the subacromial bursa vis-à-vis the outside of the supraspinatus portion. Thus a rupture of the articular parts of the aponeurosis always included the capsule, and a continued rupture including the superficial parts of the aponeurosis, always extended the wall of the bursa.

In the case of rupture of the capsule and articular parts of the aponeurosis only, the arthrogram showed a contrast-filled cavity in the deep layers of the tendon tissue near the greater tubercle. This cavity was always situated in the supraspinatus portion and, therefore, was seen in Position II. In the case of continued rupture extending into the bursa, the joint cavity communicated with the subacromial bursa through a gap in the aponeurosis. In small ruptures the gap was situated in the supraspinatus portion and only appeared in Position II, while in larger ruptures it continued into the infraspinatus portion and was visible in Position I also. When the rupture was large and inveterate, the edge of the aponeurosis was considerably retracted, and abnor-

mally thin. In a few cases, in which a pronounced trauma was lacking, the thinness of the aponeurosis was predominant in relation to the size and the degree of retraction.

Regarding arthrographic appearance and operability it was found advisable to divide the ruptures of the supraspinatus and infraspinatus portions of the aponeurosis into four groups. Cases belonging to the first two groups were almost the only ones suited for operation.

Type I. (Figs. 14—15). Ruptures in the supraspinatus portion of the aponeurosis, involving the deep layers of the aponeurosis and the joint capsule but not the floor of the bursa (14 clinical and 2 autopsy cases).

Type II. (Figs. 16—17). Ruptures situated in the supraspinatus portion of the aponeurosis, involving the capsule and the deep and superficial parts of the aponeurosis and the bursal wall and in which the aponeurosis retained its normal thickness. (26 clinical and 2 autopsy cases.)

Type III. (Figs. 18—19). Ruptures including the whole supraspinatus and infraspinatus portions, in which the retraction is so pronounced that the greater part of the superior surface of the head is exposed (7 clinical and 4 autopsy cases).

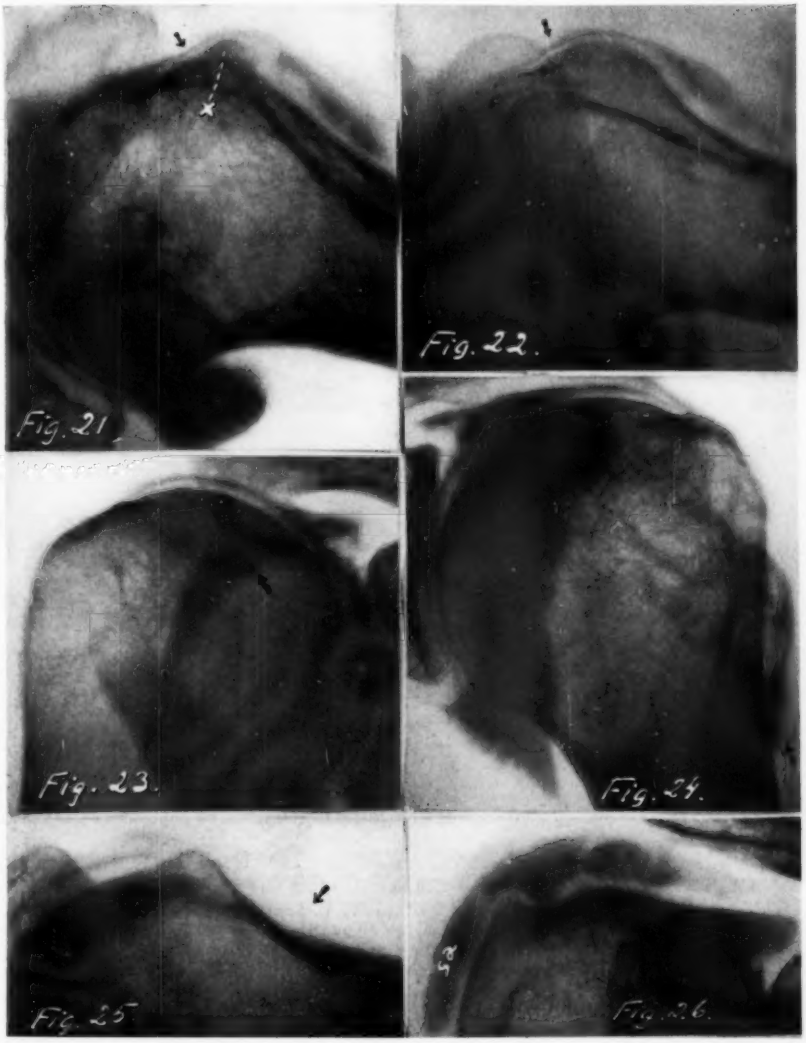
Type IV. (Fig. 20). Ruptures of the supraspinatus portion of the aponeurosis, including the capsule and bursal wall and in which the thickness of the aponeurosis is greatly reduced (4 clinical cases and 1 autopsy).

Arthrograms in ruptures of the subscapularis portion of the tendon aponeurosis

(5 clinical cases, 2 of which were operated upon, and 1 autopsy case.)

As in the supraspinatus and infraspinatus portions, the ruptures of the subscapularis portion were located near the insertion. Instead of finishing at the anatomical neck, the joint cavity stretched out over the superior part of the lesser tubercle (Figs. 21—22). The joint capsule, which in the subscapularis portion also was fixed to the distal part of the tendon, had ruptured together with the articular parts of the aponeurosis.

No case appeared to have a perforating rupture. Four clinical cases showed no contrast filling of the subcoracoid bursa, which adjoins and is attached to this part of the aponeurosis. The fifth case showed contrast filling of the subcoracoid tendon, but the fluid had obviously not entered via the subscapularis tendon, but through the perforating rupture in the supraspinatus portion into the subacromial bursa, from which it flowed into the subcoracoid bursa.



Figs. 21—22. Arthrograms in ruptures of the subscapularis portion of the tendon aponeurosis with and without osteophytes (x) on the lesser tubercle. Arrows mark ruptures.

Figs. 23—25. Arthrograms of three cases of ruptures of the long biceps tendon. In fig. 23 contrast filling at the site of the tendon. In Fig. 24 torn and irregular tendon sheath. In Fig. 25 absence of contrast filling of the tendon sheath.

Fig. 26. Arthrogram in rupture of supraspinatus portion of the tendon aponeurosis into and through an intratendinous cavity with calcified deposits.

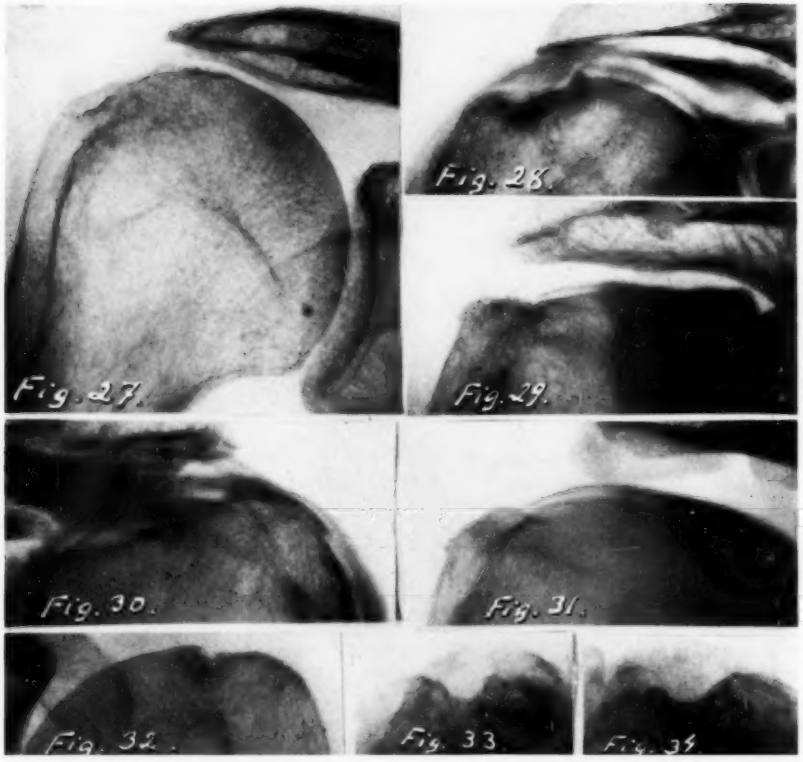


Fig. 27. Roentgenogram showing attrition of greater tubercle and acromion and subluxation of head cranially in a case of inveterate and extensive rupture of the tendon aponeurosis.

Figs. 28—31. Arthrograms illustrating changes at the tendinous insertions on the greater tubercle in cases with (28, 30) and without (29, 31) ruptures.

Fig. 32. Roentgenogram showing erosion on the anatomical neck. Arthrographically normal; at operation extra-articular cavities and no rupture.

Figs. 33—34. Roentgenograms of the intertubercular sulcus viewed axially. Picture on right shows the sulcus of healthy shoulder and picture on left shows narrowing of the sulcus of the other shoulder with a ten years old rupture of the long biceps tendon.

In one of the cases, the lesion in the subscapularis portion was accompanied by a perforating rupture in the supraspinatus portion and, in 2 other cases, by an internal rupture in the supraspinatus portion. The autopsy case and one of the clinical cases showed no sign of lesion of the supraspinatus part. Lesions of the subscapularis portion were

associated with rupture of the biceps in 2 cases. The biceps tendon in the other cases was of normal appearance.

Arthrograms in ruptures of the long biceps tendon

(11 clinical cases, 2 of which were verified operatively, and 4 autopsy cases).

In the four autopsy cases the intra-articular part of the biceps tendon was lacking and the biceps sheath was more or less completely obliterated. Instead, tendon fibres, clearly representing the long biceps tendon, issued from the inferior portion of the intertubercular sulcus. No remains of the tendon could be found at the original site of the tendon insertion by the glenoid cavity.

In the operated cases the roentgenologic diagnosis of a defect in the intra-articular part of the tendon was verified. Operation was done in these cases due to rupture in the supraspinatus part of the aponeurosis. Therefore the biceps tendon was not studied in any detail.

Six of the remaining 8 clinical cases had inveterate lesions. In the arthrogram the intra-articular part of the tendon was not visible, and the tendon sheath was not filled with contrast medium (Fig. 25). The remaining 2 cases had recent lesions and the arthrograms showed, in addition to the lack of the intra-articular portion of the tendon, a massive contrast filling of the site of the tendon (Fig. 23) and irregular cavities at the inferior end of the sheath (Fig. 24).

In only one case the lesion was confined to the biceps tendon. Two other cases also had a rupture of the subscapularis portion of the aponeurosis. The remaining cases showed lesions of the supraspinatus and infraspinatus portions.

The fraying of the biceps tendon described by MEYER could not be seen in the arthrographic cases. One of the autopsy cases showed suggested fraying, but the others were completely free from it. In this connection it should be mentioned that the thickness of the biceps tendon varied greatly in the different cases.

Other roentgenologic changes in ruptures in the tendon aponeurosis

With the exception of fractures (2 cases) and callous formations (4 cases), the following changes were observed in the ordinary roentgenograms of the shoulder in cases of ruptures of the tendon aponeurosis:

Erosion on the superior surface of the greater tubercle (Fig. 28).

Erosion on the superior surface of the anatomical neck (cf. Fig. 32).

Osteophytes on the outer and upper surface of the greater tubercle and on the adjacent articular border of the head, as well as on the lower edge of the acromion (Figs. 28, 30).

Attrition of the greater tubercle and the acromion and subluxation of the head cranially (Fig. 27).

Extensive osteophytes on the articular edges in the humeroscapular joint, and

Calcification or bone formation in the tendon aponeurosis.

Recent cases generally lacked all such changes. In the cases in which a fairly long period had elapsed between the trauma and the examination, changes were most often found on the uppermost facet of the greater tubercle, the commonest one being an osteophyte situated mainly on the outer surface of the tubercle next to the uppermost facet, often combined with small areas stretched into the territory of the anatomical neck. The free border of the acromion not infrequently showed small osteophytes. In very advanced cases the head was subluxated upwards, in addition to which the greater tubercle and the inferior surface of the acromion were often worn. Arthritic changes in the humeroscapular joint itself were uncommon. Calcified deposits or bony formations in the tendon aponeurosis were only found in exceptional cases.

Comparison with the cases without ruptures in the aponeurosis showed that the only pathognomonic sign of a tendon rupture was the attrition of the greater tubercle and the inferior surface of the acromion, combined with an upward subluxation of the head. Osteophytes on the outer surface of the greater tubercle were found more frequently in cases of rupture than others, while the remaining changes listed above appeared to be quite independent of tendon ruptures.

It may be added that in one case of inveterate rupture of the biceps tendon the intertubercular sulcus was shallowed (Figs. 33—34). The necessary projections, i. e. axial to the sulcus, were not made in the other cases.

Discussion and conclusions on the diagnostic value of the arthrography

Even at an early stage in the use of arthrography it was found that this method provided an accurate diagnosis of ruptures of the tendon aponeurosis of the shoulder joint, whereas the clinical signs were frequently misleading. Of all the cases which have so far been verified[1], only one has been wrongly diagnosed arthrographically. This was an instance of rupture in the supraspinatus portion in which the intra-

[1] One case with explorative incision and negative findings is grouped as non-verified; the incision was performed in such a way that the area of rupture could not be examined.

articular part of the biceps tendon could not be distinguished, although its tendon sheath was filled with contrast medium. It was, therefore, wrongly assumed that the biceps tendon was ruptured. Concerning the lesions of the tendon aponeurosis, the diagnosis was correct in all cases, although in one case of a minute communication between the joint and a tendinous cavity containing calcificated deposits (Fig. 26) the passage was scarcely visible.

In this material none of the clinical signs, as described by CODMAN, PALMER and others, proved to be pathognomonic. For example, there was no disturbance in the scapulo-humeral rhythm in 8 out of 47 cases, and insufficiency in abduction, or the jogging sensation when lowering an abducted arm, was lacking in 3 out of 47 cases. Furthermore, these two symptoms could not be studied in 5 other cases due to rigidity in the shoulder. The coarse, widely-spaced, soft crepitus which belongs to the picture was absent in 12 cases and could not be studied in the patients suffering from rigidity. There was no distinct tenderness over the greater tubercle in 10 cases, and no deepened groove in the greater tubercle could be felt in 10 cases. In the cases of rupture of the subscapularis portion a painful jog during abduction was the only clinical symptom. Of the cases with ruptures of the biceps tendon, the recent ones showed tenderness over the intertubercular sulcus, while in 2 of the long-standing ones the muscle belly was clearly deformed.

Thus the value of the clinical diagnosis was considerably reduced, which conforms with the information provided by CODMAN that operation revealed no rupture in 25 per cent of his cases.

On the other hand, the arthrography of the shoulder has possibilities of becoming an infallible method of detecting ruptures in the tendon aponeurosis of the shoulder joint and in the long biceps tendon. The same accuracy in the diagnosis of these ruptures cannot be reached by means of clinical methods, and ordinary roentgen examination of the shoulder is of insignificant value in these cases.

SUMMARY

The writer describes an arthrographic method of examination of the shoulder in order to study ruptures of the tendon aponeurosis or the long biceps tendon. A comparison with the clinical symptoms and the ordinary roentgenologic signs illustrates the great superiority of arthrography for correct diagnosis.

List of authors See page 577.

ZUSAMMENFASSUNG

Verf. beschreibt eine arthrographische Methode zur Schulteruntersuchung behufs des Studiums von Rupturen der Sehnenaponeurose oder der langen Bizepssehne. Ein

Vergleich mit den klinischen Symptomen und den gewöhnlichen röntgenologischen Anzeichen beleuchtet die grosse Überlegenheit der Arthrographie betreffs einer korrekten Diagnose.

RÉSUMÉ

L'auteur décrit une méthode arthographique pour examiner l'épaule en vue d'étudier les ruptures des aponévroses tendineuses ou du long tendon du biceps. Une comparaison avec les symptômes cliniques et la radiographie ordinaire illustre la grande supériorité de l'arthrographie pour un diagnostic exact.

FROM THE ROENTGEN DEPARTMENT OF ÅSÖ HOSPITAL, STOCKHOLM

N PATHOGENESIS OF RUPTURES OF THE TENDON APONEUROSIS OF THE SHOULDER JOINT[1]

by

Knut Lindblom

This study is intended to follow immediately upon the previous one, firstly because the same material has been used for both, and secondly because the observations in both studies mutually illustrate one another. The object in the present work has been to find an answer to pathogenic questions through the study of clinical and anatomic material, the main subject of inquiry being: is rupture of the aponeurosis a disease due either to wear or old age, or are one or more distinct traumas the chief cause?

From all the literature on the subject the following views may be noted in regard to the pathogenesis of ruptures of the tendon aponeurosis of the shoulder joint and of tendon ruptures in general.

CODMAN is of the opinion that one or more traumas cause the rupture, and he has mentioned that an injury leading to subcoracoid luxation is especially likely to produce a rupture of the aponeurosis. When the head of the humerus is displaced downwards the upper portion of the tendon aponeurosis is pressed against the adjacent edge of the glenoid cavity and is stretched at an angle over it. The part of the aponeurosis thereby affected depends on the rotation position of the arm. CODMAN considers that the capsule and the adjoining layers of tendinous tissue are here subjected to the greatest strain. However, he also states that age has a certain influence, seeing that the cases of rupture occur round about the age of forty or later. In his histologic examinations CODMAN has found, amongst other signs, degenerative changes in the tendon aponeurosis of older patients, but not of the younger ones, and in very aged people he found in most cases rupture of the tendinous fibers at the synovial reflection. As regards bony changes, CODMAN sometimes found an irregularity of the bone tendon line, »the blue line», and, among manual

[1] Submitted for publication Oct. 6, 1939.

workers, a thickening of the underlying bony layer, and finally, occasiona cavities in the spongiosa at the tendinous insertion.

FERGUSON has assumed that multiple small tears cause small defect in the tendon aponeurosis at the greater tubercle.

DAVIS and SULLIVAN express the view that pathologic changes du to age and type of work predispose the shoulder joint to rupture. FOWLE considers the rupture as primary to a number of pathologic processes i the shoulder.

In direct contrast to the conception that the complaint is traumati in origin are the views of MEYER, and SKINNER, who believe that it i merely a question of wear. MEYER, and SKINNER seem to have base their opinions entirely on the examination of anatomic specimens, an they undertake no explanation as to how such wear could take place. SKINNER considers that the supra- and infraspinatus tendons in new-born infants are »fleshy right up to the point of insertion», and that they lengthen out later in life.

Other opponents to CODMAN's views are the authors (TRIEPEL, FICK, MCMASTER, CRONKITE, and others) who have found, from experimental examinations, that the tensile strength of a tendon is greater than that of a muscle or bone. According to MCMASTER, the occurrence of a rupture in the tendinous tissue instead of in the bone or muscle seems to necessitate far-advanced necrosis of the tendon — such as occurs, for example, after a period of ligaturing — or a defect in half of the tendon. However, a previous trauma can damage the tendinous tissue to such an extent that a tendon rupture may later occur, as HONIGSMANN and others have shown in connection with the extensor pollicis longus.

From a pathogenic point of view the reports on disturbances in the vascular system of the tendinous tissue are also interesting. RAU has found that the tendinous tissue in people over the age of 25 to 40 is considerably poorer in blood vessels than that in adolescents. SANDSTRÖM and WAHLGREN believe that a local ischaemia is a contributory factor in the appearance of calcified deposits in the tendinous tissue.

Briefly, we may say of all these divergent statements in the literature, that they do not give any clear picture of the pathogenesis and that to a large extent they are contradictory.

The Author's Investigations

The investigations were concerned, in the first place, with anatomic changes, that is, changes in the structure of the tendon aponeurosis and its vascular system, and also changes in the tensile strength of the tendon aponeurosis when strain was applied in the direction of the fibers, firstly parallel with the superior surface of the head of the humerus, and sec-

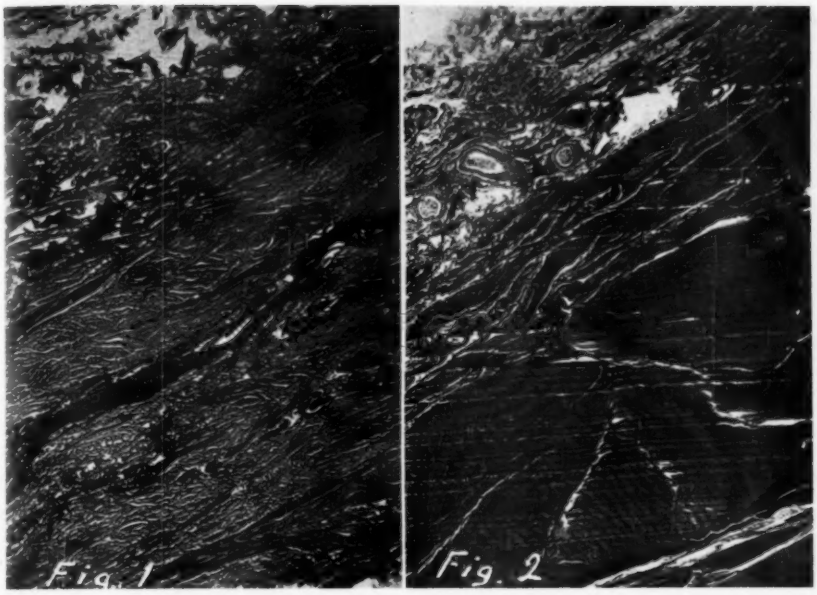
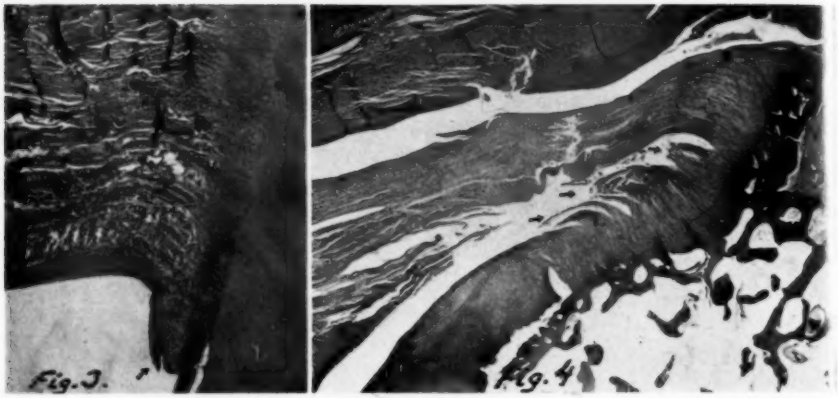


Fig. 1—2. Microphotographs of cross sections of supraspinatus portions of the tendon aponeurosis of two cases, the left a man of 26, the right a man of 68. Note the difference in homogeneity.

ondly at right angles to the tendinous insertion. In the second place, the object of the investigations was to search for facts in the clinical material that might throw light on pathogenic questions. The *material* was the same as in the previous roentgenologic study.

Changes in the Structure of the Tendon Aponeurosis. For this purpose 36 anatomic specimens were examined, 8 of which were from children up to the age of 2, and the others from adults ranging in age from 26 to 86. Rupture of the aponeurosis was present in 9 of the cases between the ages of 32 and 86. All the specimens were examined histologically, for which purpose large slices were cut off, including both the tendinous tissue and the bone at the tendinous insertion. Before this, the specimens had been studied macroscopically, careful note being made, amongst other things, of the distance between the junction of the muscle and tendon and the tendinous insertion, and also of the width of the head of the humerus. In addition to these 36 anatomic specimens, a microscopic examination was made of tissue that had been resected from the edges of ruptures found in 14 patients who had been operated on.



Figs. 3—4. Microphotographs of longitudinal sections of supraspinatus portion of the tendon aponeurosis of two cases. Left figure from a new-born child shows a normal process of connective tissue at the cartilage-tendon angle. Right figure shows processes due to rupture of the internal parts of the aponeurosis.

It appeared from the microscopic examination of the anatomic specimens that in nearly all the older persons, irrespective of their work or sex, the tendinous fascicles were collected into a more homogeneous, sclerotic tissue (Fig. 2), while in the younger persons narrow, tendinous fascicles were separated by streaks of looser tissue (Fig. 1). In the older persons also, there were often — especially in the centre of the fascicles — small nests showing swelling of the cells of the connective tissue and altered staining reaction, which was considered to indicate fibrinoid degeneration. In the rupture cases these degenerative changes were found again also in the edges of the rupture, but as a rule they were so definitely confined to these that it seemed more likely that the degeneration was secondary to the rupture and not the real cause of it.

The specimens taken from the patients operated on showed without exception the above-mentioned changes in the appearance of the cells and the staining reaction of the fibrils. As none of the patients was operated on earlier than four weeks after the trauma, it was impossible to establish whether the changes were primary or secondary to the rupture.

As regards the tendinous insertion, the histologic examination of the anatomic specimens from older persons often showed a bursting through of the bone line, as a result of which the marrow processes had grown into the tendinous tissue (Fig. 11). A bony formation was sometimes found in the tendinous tissue, and in exceptional cases there were amor

hous calcified deposits chiefly localized in the outer portions of the endinous tissue. The intruding marrow processes had no connection vith ruptures in the tendinous tissue, either as regards frequency or ocalization, since they appeared just as often in non-ruptured tendinous issue, and more often on a level with parts of the tendon that were unhanged. In no case had the tendon ruptured as far down as the bone ine. The bony formations and calcified deposits also had no connection t all with ruptures, as far as localization was concerned.

The comparison between the length of the tendinous tissue and the idth of the head showed the relation, in fetuses and children, of 9 : 14 = .64 (8 cases), and in adults, of 73 : 131 = 0.56 (27 cases). In one case ith a very extensive rupture the length of the tendon could not be letermined and the case was excluded. The number of cases is not ufficient to permit definite conclusions, but the result of the comparison hows that there is no support for SKINNER's assumption that the length d the tendon increases with age.

One point must be stressed here in connection with the result of the microscopic examinations. CODMAN has described in his monograph of 1934, a small process of connective tissue, situated in the angle between the articular cartilage and the tendinous insertion, as being part of the ruptured tendinous tissue. In our material, however, this connective tissue was present in every case, including the children (Fig. 3). Sometimes it did not slide freely into the joint but followed the cartilage edge and outlined it. In one case, large villous proliferations extended from this area, but neither in this nor in the other »normal» cases was there any proximal fragment of tendinous tissue corresponding to this connective process, as was the case in a genuine tendon rupture (Fig. 4).

In no case were the changes such as would indicate a gradual attrition of the aponeurosis as the cause of a rupture. Only in very advanced stages of rupture were changes found which could be partly interpreted as wear.

Calcified deposits in the tendon aponeurosis were only observed in one of the anatomic specimens, and these were inconsiderable and situated in the outer portions of the aponeurosis. Inflammatory changes were totally absent.

Changes in the Vascular System of the Tendon Aponeurosis. The vascular system of the tendon aponeurosis was studied in 12 of the 36 anatomic specimens. The arteries of the shoulder and arm had been filled with cinnabar gelatine from the subclavian artery. Two cases had a rupture in the supraspinatus portion. Various ages were represented, but the object of the examinations was not so much to compare the vascular system at different ages as to compare the vascular system of different parts of the aponeurosis.

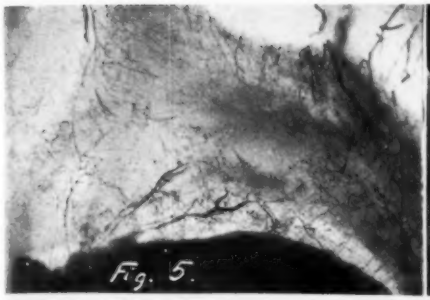
The examination of the specimens was done by means of roentgeno graphs of various parts of the tendon aponeurosis, taken first »en face« and then, after being cut in slices a few millimeters thick, in profile.

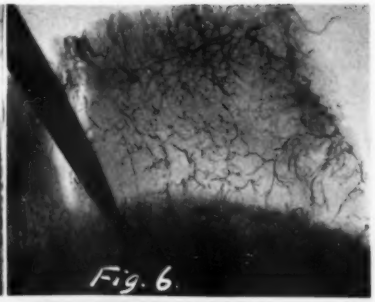
The pictures taken »en face» showed a somewhat similar vasculariza tion in two of the cases, namely, a fetus and a 68 year old man, and the others showed slightly fewer vessels in the peripheral area of th supraspinatus than in the subscapularis- and infraspinatus portions Apart from the cases with rupture in the supraspinatus portion of th aponeurosis, this difference was most pronounced in a 26 year old man But in spite of this, the last-mentioned case showed no histologic sign of degeneration in the supraspinatus portion of the tendon aponeurosis In fact, there was on the whole no clear parallelism between the vascula system and the histologic signs of degeneration.

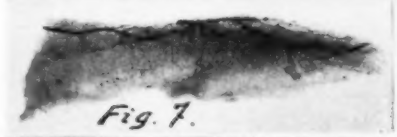
In most of the specimens the profile pictures showed less rich vas cularization in the articular portions of the aponeurosis than in the external layers. A closer examination showed, however, that the difference was only a seeming one, as the outer layers contained in addition to their own vessels, the larger arteries afferent to both the outer and the inner layers. At the tendon insertion, however, especially in the supraspinatus portion, there were areas with only a small number of vessels or wholly without them. But these were not predilection areas for degenerative changes. In exceptional cases such areas were vascularized partly from the vessels of the marrow bone (Fig. 11). In one of these there was a small haematoma, but in the others the histologic picture showed nothing noteworthy.

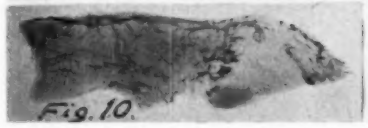
A comparison with the conditions in the long biceps tendon proved interesting. (Figs. 12—13). Its vessels came partly from the tendon insertion and partly from the muscular end. An area about 2 cm. from the tendon insertion appeared to lack vessels, and in the articular portion of the rest of the tendon the vessels were situated mostly in the outer portions that face away from the joint. With one exception, definite degenerative changes were absent in both the rich and poor vascular systems. The exception was the one case with a rupture in the supraspinatus portion of the aponeurosis, where the biceps tendon showed a thickening, with histologic signs of degeneration, situated in the defective area of the tendon aponeurosis.

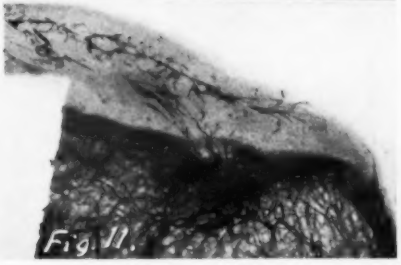
Changes in the Tensility of the Tendon Aponeurosis against Strain applied in the direction of the Fibers. The examination of the tensility of the tendon aponeurosis was made chiefly with the object of ascertaining whether it was possible, without such a weakening of the tendinous tissue as would be caused by severing half of its fibers, to produce a rupture in the tendinous tissue instead of in the bone or muscle. The reports in the literature did not support this theory, and when strain was applied



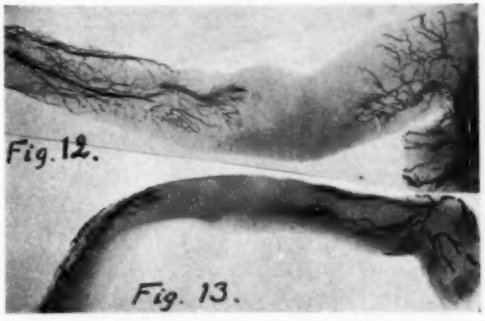








Figs. 5—11. Roentgenograms of supraspinatus portions of the tendon aponeurosis of four cases. The arteries are filled with cinnabar gelatine. In Figs. 5—6 the aponeurosis is seen en face, in Figs. 7—11 in profile. Figs, 5 and 7 represent a man of 26 without rupture or degenerative changes. Figs. 6, 8, and 11 represent a man of 68 without rupture but with some histological signs of degeneration. Fig. 9 represents a woman of 32 without rupture or histologic changes. Fig. 10 represents a woman of 32 with rupture in an adjacent part (See Fig. 4) and with histologic signs of degeneration. Note the abscence of any rule for the occurrence of vascular changes.



Figs. 12—13. Roentgenograms en face and in profile of the biceps tendon. See text.

Figs. 16—18. Microphotographs of longitudinal sections of supraspinatus portions of the tendon aponeurosis of two cases. The specimens were fixed in formalin in abduction with the strain applied (See text) and embedded in celloidin. Fig. 16 represent a new-born child. Figs. 17—18 represent left and right shoulder of a man of 40, the latter with experimental rupture. Note the difference in tension of the fascicles.

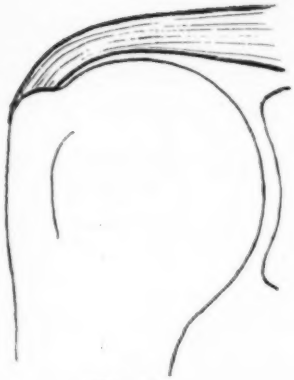


Fig. 14.

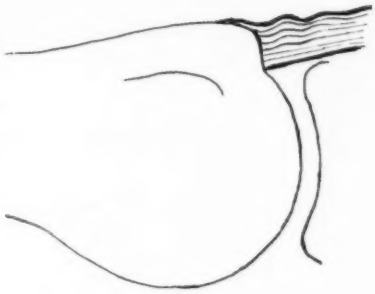


Fig. 15.

Figs. 14—15. Sketches of the humero-scapular joint in adduction and abduction illustrating the difference in tension of the fascicles of the tendon aponeurosis in abduction.

in the direction of the fibers, with the aponeurosis lying towards the head (Fig. 14), a rupture occured without exception in the muscle tissue. On the other hand, when strain was applied at right angles to the tendinous insertion (Fig. 15), it was possible to produce a rupture in the tendinous tissue, and the experiments made in connection with this will be described in the following paragraphs.

In 7 specimens a 1—1½ cm. wide portion of the supraspinatus tendon was isolated from other parts of the aponeurosis by parallel incisions running out to the greater tubercle. The incisions went up into the muscle and down into the bone. The muscular end was sutured in several places, half in the deep and half in the outer portion. The specimen was then subjected to a strain in the lengthwise direction of the tendinous fibers, not along the upper surface of the head but at right angles to the superior facet of the greater tubercle. Before the real strain was applied the sutures were knotted together so that they were evenly stretched in the direction just mentioned. In a first series of experiments the strain was applied slowly, in a later series more rapidly.

In this way, when the strain was slow, it proved possible, in children, to stretch both the inner (= articular) and outer parts of the tendon. On the other hand, in older persons, the inner parts were stretched while the outer ones remained wrinkled. The difference is demonstrated by the specimens, illustrated in Figs. 16—18, that have become fixed in formalin during the pulling process.

In the experiments when the strain was rapidly applied, the method was as follows: a weight of 4—15 kilograms was fastened at the point

of juncture of the sutures. The aggregate of humerus, tendons, sutures and weight were brought into a vertical position with the weight resting on the floor. The humerus was then jerked upwards so strongly that the weight left its support for a moment. The result can be seen from the following table:

Age	Work	Side	Weight	Number of Jerks	Result
0 yrs . . .	—	right	4 kg.	2	A break at the sutures.
0 » . . .	—	left	4 »	1	Ditto.
2 » . . .	—	right	6 »	4	Ditto.
40 » . . .	Engineer	right	15 »	1	Rupture of the inner = articular portions of the tendon at the greater tubercle (Fig. 18).
58 » . . .	Parquetry Floor Layer	right	8 »	3	Ditto.
70 » . . .	Author	right	11 »	3	After first jerk rupture of inner portions, then complete rupture.
70 » . . .	Author	left	11 »	4	Ditto.

When the tendons were examined microscopically it was found that those in the 4th to 7th experiments showed no degenerative changes at the point of rupture. The tendons in the first three experiments showed no degenerative changes at all. In none of the specimens was there any sign of injury in the bone.

Clinical Data Illustrative of Pathogenesis. The following aspects were studied in the clinical material with and without ruptures in the tendon aponeurosis, namely, age of life, character of the occupation, sex, relation between age of life and size of rupture, localization of rupture in the right or left shoulder in right- or left-handed people, the presence of traumas and the nature of the trauma, and finally, the presence of other injuries caused by the accident.

It should be noted that the material underwent selection right at the beginning, cases under 30 years of age not being arthrographed to the same extent as the others. The reason for this was our knowledge of the slight prospect of finding a tendon rupture in young people. However, the indication for arthrography was for the most part the same, namely, a suspected rupture, both in cases with and without rupture.

The Age of Life, in cases both with and without rupture, is demonstrated in a diagram. In the former group the age was to a great extent considerably lower than in the latter, but within the age limits of 55—65 years the number of cases without rupture constantly amounted to one-third of those with rupture. Between the ages of 45—55, the number of cases was the same in both groups. The decrease after the age of 65 is explained by the fact that some of the patients had reached the age of

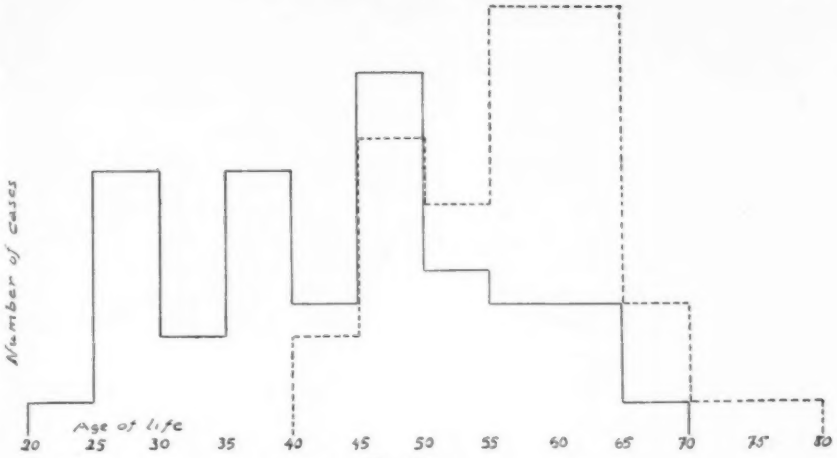


Fig. 19.

——— = cases without rupture, - - - - - - - = cases with rupture.

retirement; most of the cases, in fact, gave accidents at work as the cause of the trouble.

Character of Occupation. In the majority of the cases this was, or had been, some form of manual labor, and in this respect there was no difference between the cases with and without rupture. Exceptions were met with in both groups. In six of the ruptures the occupation had not been manual labor; these were a managing director, an overseer (bilateral rupture), a married woman, and two spinsters. In five other cases the patient's occupation did not imply manual labor; these were a taxi-driver, a policeman, a painter, a cook, a tobacco factory worker.

Sex. Five cases were women and forty-six men. It should be mentioned here that the youngest case in the series of anatomic specimens was a 32 years old working-class wife. The specimen can be seen in Fig. 4. Unfortunately, it was not possible to obtain information as to whether a trauma had occurred or not.

Relation between Age of Life and the Size of the Rupture. The average age in cases with a widely diffused rupture was fifty-five (11 cases), and in cases with a rupture in the supraspinatus portion only, fifty-four (42 cases). In the former group the history of the illness was of seven years' duration, and in the latter, $1^1/_2$ years, counting from the first trauma reported.

Rupture in the Aponeurosis of the Right or Left Shoulder in Right- or Left-Handed people. The rupture was on the left side in 15 cases and on the right in 37 cases. In the former group without exception the

patient in question was right-handed. In the latter, reports were missin in 8 cases while in the others the patients were right-handed. Ther was a bilateral rupture in 3 cases, two of which showed larger ruptur on the left than on the right side. Bilateral arthrography was used i two of the cases with rupture on the left side, although clinical sympton from the right side were not present; the right side was then intact. Ir versely, bilateral arthrography was used in one of the cases with ruptu on the right side without symptoms on the left side being present, th last-mentioned side was then intact.

Traumas. A probable trauma was present in 45 cases and was lackin in 5 cases. In 2 cases reports were uncertain, while in several others lateral haematoma appeared near the biceps. The nature of the traum was given, or appeared, as follows:

a) In 22 cases a fall on the hand or elbow, with an abducted arm.
b) » 16 » a fall »on the shoulder» or upper arm.
c) » 10 » the abducted arm had been wrenched caudally.
d) » 3 » a fall through a hatch, recovery at the edge of th hatch, and resulting hyper-extension of the arm.
e) » 2 » a violent jerk without external injury.
f) » 2 » no possibility to reconstruct the course of the accident.

In the above are included 9 cases with two to three traumas. Of the cases with rupture of the subscapularis two belonged to groups b) and f), while two other cases, with combined ruptures in the supraspinatus- and subscapularis portions and the long biceps tendon, belonged to groups a) and d) respectively. In the cases with no known traumas, two were carters, and two suffered from chronic polyarthritis with severe destruction of the joint; a fifth case was a 46 years old worker in a tobacco factory, doing no heavy manual work.

Presence of Other Injuries Caused by the Accident. In the cases with rupture a definite luxation occurred in connection with the accident; in 3 cases (52, 65 and 60 years of age), two inferior and one posterior respectively; in two more cases the statements in regard to luxation were uncertain. One case showed a longitudinal fissure in the greater tubercle, one an avulsion fracture of the upper part of the crista tuberculi minoris, one a fracture of the radius, one a fracture of the metacarpal bone, four cases callous formation at the greater or lesser tubercle and two cases deformity of the muscle belly of the biceps appearing immediately after the accident. By way of comparison, it should be noted that among the cases without rupture in the aponeurosis luxation was present in 4 cases (20, 26, 47 and 48 years of age), all of these being an inferior luxation, and a partial attrition of the greater tubercle in 3 cases (25, 32 and 51 years of age).

Non-traumatic Changes in the Tendon Aponeurosis. These were represented by 2 cases of highly destructive chronic polyarthritis and by 1 case of large intra-tendinous calcified deposits occupying nearly the whole thickness of the aponeurosis.

Discussion and Conclusions

It appears from our study of the clinical and anatomic material that as a rule the appearance of a rupture in the tendon aponeurosis of the shoulder joint seems to require more than one condition.

The clinical material shows that a rupture does not occur unless the patient has reached a certain age (30 to 40 years). One absolute requirement, therefore, is a change in the tendinous tissue due to age. The anatomic studies indicate that this change due to age consists of a sclerosization of the tendinous tissue.

It also appears from the clinical material that a definitely introductory trauma — except for a few cases with strongly destructive processes — is an almost constant occurrence in the clinical picture. We are not entitled to postulate from this that traumas or destructive processes are necessary conditions for the appearance of a rupture, but the following facts may be regarded as direct or indirect support for the existence and significance of traumas:

1) In cases of rupture, luxation or signs of a bony lesion occurred not seldom as a result of the accident.

2) In the cases of rupture where arthrography was used on the undamaged shoulder as well, rupture was found to be absent in the healthy shoulder.

3) In cases of rupture with traumas only in the left shoulder, the rupture was not present in the right shoulder, in spite of the fact that the patients were right-handed.

4) Under conditions resembling those of the intravital state it was possible experimentally to produce in the anatomic specimens a rupture of the tendon aponeurosis of the shoulder, and such a rupture did not differ in appearance from one occurring during life.

The experiments demonstrated still another condition for the appearance of ruptures, namely, that as far as ruptures in the supraspinatus are concerned, the injury must occur or affect the arm in an abducted or externally rotating position. Analogously, ruptures of the subscapularis should therefore require an internally rotating position of the arm. There is no definite support for this theory to be found from the clinical material, some of the patients with ruptures in the supraspinatus portion of the aponeurosis having stated that they fell on the shoulder or upper arm, i. e. on an arm that had already been adducted. However, there are

many difficulties in reconstructing in detail the course of the accident, and the observations made from the clinical material could therefore not be conclusive on this point. In most instances, an abduction probably occurred during the fall as a protective measure. It should be emphasized that falls on the hand or elbow because of elevation of the shoulder are followed by a forced adduction in the scapulo-humeral joint.

Finally, the experimental investigations show that a rupture of the tendon aponeurosis can, and very often does, take place in stages. This view is supported by the fact that cases with non-perforating ruptures comprise a great deal of the clinical material.

There is nothing in the study of either the clinical or anatomic material which may give rise to the assumption that ruptures of the tendon aponeurosis arise through wear. Histologically, there were no signs of this; also there was no relationship between the age of life and the size of the rupture, and as regards the character of the occupation there were far too many exceptions from the rule that the complaint affects manual works. The combination of right-handedness and ruptures on the left side only is also an argument against the theory of wear.

The study of the tendon aponeurosis from the point of view of vascular changes gave no unanimous conclusion. The usual histologic signs of degeneration in the tendinous tissue could not be proved as being primary to the rupture.

SUMMARY

The studies of clinical and anatomic material and the experimental investigations on anatomic specimens led to the following conclusions:

Ruptures in the tendon aponeurosis of the shoulder joint require as a condition for their appearance a certain change in the tendinous tissue due to age, and usually, in addition, one or more distinct traumas or, in exceptional cases, a strongly destructive process.

Ruptures in the tendon aponeurosis of the shoulder joint do not arise through attrition of the aponeurosis.

ZUSAMMENFASSUNG

Die Studien an klinischem and anatomischem Material und die experimentellen Untersuchungen anatomischer Präparate führten zu nachstehenden Schlussfolgerungen:

Als Bedingung für das Entstehen einer Ruptur der Sehnenaponeurose des Schultergelenks bedarf es einer gewissen Altersveränderung des Sehnengewebes und gewöhnlich ausserdem eines ausgesprochenen Traumas oder mehrerer oder in Ausnahmefällen eines stark destruktiven Prozesses.

Rupturen in der Sehnenaponeurose des Schultergelenks entstehen nicht durch die Abgenutztheit der Aponeurosis.

RÉSUMÉ

L'étude du matériel clinique et anatomique, jointe à des recherches expérimentales sur pièces anatomiques, permet les conclusions suivantes.

Les ruptures des aponévroses tendineuses de l'articulation de l'épaule n'apparaissent qu'à condition qu'il y ait une certaine altération sénile du tissu tendineux, et qu'également dans la plupart des cas un ou plusieurs traumas distincts, ou dans des cas exceptionnels un processus fortement destructif, aient précédé.

Les ruptures des aponévroses de l'articulation de l'épaule n'apparaissent pas à la suite de l'usure de l'aponévrose.

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AUS DER RÖNTGENDIAGNOSTISCHEN ABTEILUNG DES KRANKENHAUSES DER UNIVERSITÄT LUND, SCHWEDEN (CHEF: DOZENT HANS HELLMER)

SPONTANES GASPYELOGRAMM[1]

von

Olle Olsson

Seit langem weiss man aus dem klinischen Symptom der Pneumaturie und bestimmten Befunden bei der Obduktion, dass in den Urinwegen bei gewissen pathologischen Zuständen Gase vorhanden sein können. Eine derartige Pneumaturie, wie sie zuerst von RACIBORSKY nach einer Beschreibung in der naturwissenchaftlichen Literatur aus dem Jahre 1671 mitgeteilt wurde, kann sehr verschiedene Ursachen haben. Sie kommt unter Umständen dadurch zustande, dass Gase, meist Luft, beim Katheterisieren, Cystoskopieren oder anderen ähnlichen Manipulationen in die Blase gelangten. Nach Elektrokoagulation in der Blase können ebenfalls durch den Gewebszerfall Gase entstehen. (HAMBLETON-LACKEY-VAN DUZEN zeigten, dass diese Gase mit von aussen kommendem Sauerstoff eine explosive Mischung ergeben können). Ferner bilden Fisteln, spontane oder operative, zwischen Ureter-Blase-Urethra und Darm, oder auch Urinwegsbakterien die Ursache einer Gasentwicklung. Eine Pneumaturie auf Grund von Bakterien ist am häufigsten bei Diabetikern mit einer Cystitis. Bei diesen kommt es zu Gärungsprozessen durch Hefepilze, die eine Alkoholgärung unter Kohlensäureentwicklung hervorrufen, oder durch Colibazillen, die ausser der Kohlensäure noch Wasserstoff bilden. Aber auch in zuckerfreiem Harn können Gase entstehen, nämlich bei vorhandenem Eiweiss, durch Coli oder den Bazillus aerogenes lactis (ADRIAN & HAMM). Eine weitere Form bakterieller Gärung ist die Schwefelwasserstoffbildung aus den Urinsulfaten durch Coli (SENATOR). Diese sogenannte Hydrothionurie geht jedoch nicht immer mit einer Pneumaturie einher. Ein Fall mit Gasentwicklung in der Blase durch Gasbrandbazillen infolge eines Granatschadens wurde von PFLAUMER mitgeteilt. Dieser wies jedoch kein Emphysem der Blasenwand auf, und die Gase hatten sich wahrscheinlich aus dem Blut in der Blase gebildet. WEISER veröffentlichte einen Fall mit starker Pneumaturie bei einer Gasphlegmone der Blase durch den Bazillus phlegmones emphysematosae

[1] Bei der Red. am 9. X. 1939 eingegangen.

Fränkel-Welch. Ein Fall, bei dem röntgenologisch Luft in der Blase festgestellt wurde, ist von MULSOW & GILLIES beschrieben worden. Es war eine Cystitis, aber ohne gasbildende Bakterien vorhanden. Der Harn enthielt weder Eiweiss noch Zucker, reagierte aber stark alkalisch. Die Verff. nehmen an, dass sich die Gase vielleicht durch eine Reaktion des sauren Nierenurins mit den Carbonaten des Retentionsharnes der Blase gebildet haben. Sie halten es jedoch für wahrscheinlicher, dass gasbildende Bakterien vorhanden gewesen waren, die in dem alkalischen Blasenurin zugrunde gingen.

Bei allen diesen Fällen war Gas in der Blase vorhanden. Gase in den oberen Urinwegen wurden bei der Obduktion einmal von O. ZUCKERKANDL und einmal von SØRENSEN beschrieben.

Röntgenologisch ist der Befund von Gasen in der Blase keine Besonderheit. Seitdem JACHES & FURNISS in den ersten Jahren der Pyelographie den Gebrauch, von Sauerstoff als Kontrastmittel empfahlen, ist das Vorhandensein von Gasen in den oberen Urinwegen für den Röntgenologen ein bekanntes Bild. Soweit ich aus der Literatur ersehen konnte, wurde jedoch nur einmal die Röntgendiagnose einer spontanen Gasfüllung der Harnwege mitgeteilt. (Schaukasten-Notiz von NOGUEIRA in der Zeitschrift für Urologie *29*, 1935 S. 275.) NOGUEIRA zeigt ein Übersichtsbild der Urinwege mit Gas im linken Nierenbecken und Ureter, und in der Blase. Die Ursache des Befundes konnte nicht geklärt werden.[1]

Im Folgenden soll über einen Fall berichtet werden, bei dem der röntgenologische Befund von Gasen in den Urinwegen nicht nur die morphologische Diagnose einer Pyelonephritis mit Papillennekrosen ergab, sondern dazu auch die bakteriologische Ursache erschliessen liess.

69-jährige Frau, die seit dem Jahre 1933 zweimal wegen eines Coma diabeticum in der Medizinischen Klinik behandelt worden war. Diät und Insulin. Einlieferung in die Med. Klinik am 20. 4. 39 nachdem sie am Tage vorher mit Schüttelfrösten und allgemeinen Krankheitserscheinungen ohne Lokalsymptome erkrankt war.

Status präsens: Kleine, blasse Frau, die apathisch im Bett liegt. Kein Acetongeruch. Temp.: 38.9.

Bei der Palpation des Bauches, besonders dessen oberen Teiles, spannt die Pat. und äussert Schmerzen. Der Unterbauch ist weich und unempfindlich. Resistenz unter dem linken Rippenbogen herunter bis zu Nabelhöhe. Perkutorisch Dämpfung über diesem Gebiet. Schmerzhaftigkeit bei Palpation der linken Nierenloge.

Rectal und vaginal kein besonderer Befund.

S. R. 56/90. Reststickstoff 73 mg %.

Urin: strohgelb, trübe, sauer. Sediment: Einzelne rote Blutkörperchen, 2—5 weisse, 0—5 Epithelien pro Gesichtsfeld.

Röntgenuntersuchung am 22. 4. 39: Auf einem Übersichtsbild des Bauches beträgt der rechte Nierenschatten 13 × 6 cm, während der linke $15^1/_2$ × 9 cm. misst. Die Fettlinien

[1] In »Nordisk Medicin» Nr 42, 21. X. 1939 hat Jensen einen Fall von Pneumopyonephrose mit Rtg-Befund von einer Gasansammlung im rechten Nierenbecken geschildert.

treten links wesentlich schlechter hervor, und auch die Psoaskonturen sind auf dieser Seite undeutlicher.

Mit der Diagnose Diabetes mellitus + Pyonephrosis sin. wird die Patientin am 22. 4. 39 auf die Chirurgische Abteilung verlegt.

Cystoskopie unmittelbar. (KOCH): *Blasenschleimhaut:* Mässige Trigonuminjektion. — *Rechte Ureterenmündung:* o. B. — Klarer Urin mit normaler Tropfgeschwindigkeit. — *Linke Ureterenmündung:* aus der Öffnung wird dicker Eiter ähnlich kleinen Würmern entleert. Der Ureter ist leicht und in ganzer Länge zu katheterisieren; keine Entleerung durch den Katheter.

Zwei Tage später erneute *Cystoskopie* (PETRÉN): Der linke Ureter wird katheterisiert, aber nicht in ganzer Länge. Nach einer Spülung tropft langsam eine Flüssigkeit heraus, die hauptsächlich aus der eingespritzten, wenn auch mit einer gewissen Harnfarbe besteht. Mikr.: Massenhaft weisse Blutkörper, zahlreiche rote, wenig Epithel. Kurze coliähnliche Stäbe.

Röntgenuntersuchung am 16. 5. 39: Die linke Niere ist etwas kleiner als bei der vorigen Untersuchung am 22. 4. 39. Keine Konkremente in den Harnwegen.

Wegen des Diabetes wird die Patientin wieder auf die Medizinische Abteilung verlegt. Stabilisierung des Diabetes so gut als möglich. Die Infektion der Harnwege macht jedoch immer stärkere Beschwerden. Temp. bis 38.6.

Wiederholung der *Cystoskopie* am 12. 7. 39 (MILLBOURN) ergibt im Wesentlichen dasselbe Resultat wie die vorhergehenden Untersuchungen.

Am 18. 7. 39 (6 Tage nach der letzten Cystoskopie) erneute *Röntgenuntersuchung*. Auf einem Übersichtsbild der Urinwege beträgt der rechte Nierenschatten 10 × 7 cm., der linke 11.5 × 6 cm. Seit der Untersuchung am 16. 5. 39 ist der linke Nierenschatten also wesentlich kleiner geworden. *In dem linken Nierenschatten sind multiple Gasansammlungen festzustellen, die im Nierenbecken, und als gut erbsengrosse, rundliche Hohlräume zu einer Kante 1 cm innerhalb der äusseren Nierenkontur angeordnet sind.* (Abb. 1.) Die letzteren Gasansammlungen behalten ihre Lage auch bei Untersuchung im Stehen und in Seitenlage. Wenig Gas in der Blase. — Bei der Urographie kommt es rechts zur normalen Zeit zu einer normalen Ausscheidung. *Links ist die Ausscheidung etwas verzögert und auffallend gering. Eine Füllung der Calyces und des Nierenbeckens kommt jedoch zustande, und es ist nun deutlich zu sehen, dass die oben beschriebenen gasgefüllten Hohlräume direkt vor jedem Calyx liegen und von Destruktionen in den Nierenpapillen zeugen.* (Abb. 2.) — Aus der Röntgenuntersuchung geht also hervor, dass die Funktion der linken Niere herabgesetzt ist, und multiple Papillennekrosen und Gase in der Niere und den Harnwegen vorhanden sind. — *Diagnose: Pyelonephritis mit Papillennekrosen, verursacht durch gasbildende Bakterien, wahrscheinlich Coli.*

Die bakteriologische Untersuchung des Blasenurins ergab Coli.

Der Zustand der Patientin verschlechterte sich jedoch immer mehr, und es kam unter zunehmendem Marasmus am 22. 8. 39 zum Exitus.

Bei der *Obduktion* (WINBLAD) wurde die röntgenologische Diagnose vollständig bestätigt, und eine sorgfältige bakteriologische Untersuchung (NYMAN) ergab mit voller Sicherheit, dass Coli die gasbildenden Bakterien gewesen waren.

Auszug aus dem Obduktionsprotokoll 365/1939:

Klinische Diagnose: Diabetes mellitus + Cystopyelitis + freie Gase im linken Nierenbecken.

Abb. 1.

Pathologisch-anatomische Diagnose: Diabetes mellitus + Pyelonephritis e bacill. coli mit Gasbildung in den Nierenpapillen durch Gärung des Urinzuckers + Cardiosclerosis + Bronchitis purulenta.

Entwicklung von Leichenphänomenen.

Das linke Nierenbecken und der Ureter werden unter Wasser geöffnet. Dabei steigen einige Luftblasen auf. Die linke Niere weist keine Para- oder Perinephritis auf. Die Nierenoberfläche ist fest und zeigt zahlreiche kleine Blutungen. Mässige Erweiterung des Nierenbeckens. An einzelnen Stellen, besonders in den kleinen Calyces kleine weiche gelbbraune Klumpen. Nekrosen an den Papillenspitzen. (Abb. 3.) Das Nierenparenchym ist gelb und verwaschen. Die rechte Niere besitzt auch im Becken derartige kleine weiche Konkremente. Hier keine Zeichen einer Glomerulo-

Abb. 2.

oder Pyelonephritis. In der Blase ebenfalls freies Gas. Geringe Injektion um die linke Uretermündung. Sonst keine Anzeichen einer Infektion.

Mikroskopische Untersuchung:

Die Papillenspitzen der Nieren zeigen Nekrosen und heftige entzündliche Infiltration unspezifischer Art. Vereinzelt reicht die entzündliche Infiltration strichförmig herauf in das Parenchym, und hier ist auch ein Ausfall der Glomeruli vorhanden. Das histologische Bild zeugt von einer unspezifischen Pyelonephritis.

Bakteriologische Untersuchung:

Material des linken Nierenbeckens. Züchtung: Das Material wurde in physiologischer Kochsalzlösung aufgeschlemmt und von dieser Aufschlemmung dann auf verschiedenen Substraten aerob und anaerob

Abb. 3. Sektionspräparat. Ur = Ureter, Pn = Papillnekrose.

gezüchtet und Meerschweinchen intramuskulär gespritzt. Ausserdem wurde ein Direktausstrich gemacht und nach Gram gefärbt.

Resultat: Das Direktpräparat zeigte ausschliesslich Gr. — Stäbchen, hauptsächlich Coli. Aerobe Züchtung: Bouillon, Trouche: Gr. — Stäbchen. Gassner, Conradi: Coli. Blutagar: Coli. Peptonwasser: Indolprobe positiv nach 24 Stunden. Gärungsprobe: Glukose, Lactose und Mannit ergaben sowohl Gas wie Säure nach 24 Stunden. Gelatine: Keine Verflüssigung. Glukoseagar: Gasbildung. Beweglichkeit: +

Anaerobe Züchtung: Ein Teil der Probe wurde auf dem Wasserbade von 80° während $^1/_2$ Stunde erhitzt. Von dieser Probe wurde auf Ascites-Bouillon und Blutagar in einer Anaerob-Glocke und auf Cysteinagar gezüchtet. Resultat: 0 Wachstum.

Ein anderer Teil der Probe wurde unbehandelt anaerob gezüchtet. Resultat: Nur Gr. — Stäbchen.

Meerschweinchenprobe: 1 ccm der Aufschlemmung wurde am Meerschweinchen intramuskulär injiziert. Resultat: Nach 7 Tagen noch keine lokale oder allgemeine Reaktion.

ZUSAMMENFASSUNG

Bericht über einen Fall, bei dem röntgenologisch eine Pyelonephritis e bacill. col mit Papillennekrosen durch ein Gaspyelogramm festgestellt wurde, das sich bei eine älteren Diabetikerin spontan durch bakterielle Vergärung des Harnzuckers gebildet hatte. Die Röntgendiagnose wurde durch die Sektion in allen Einzelheiten vollkommen bestätigt

SUMMARY

The author reports a case in which a pyelonephritis of colibacillary origin with papillary necrosis, in an elderly female diabetic, was diagnosed roentgenologically by means of a gas pyelogram; the gas having formed spontaneously as the result of fermentation of the sugar in the urine. At necropsy, the roentgenological diagnosis was thoroughly confirmed.

RÉSUMÉ

Exposé d'un cas dans lequel une pyélo-néphrite e bacill. coli avec nécrose papillaire fut découverte à l'examen radiologique, à l'aide d'une pyélogramme au gaz. Il s'agit d'une vieille diabétique qui fit une fermentation bactérienne spontanée du sucre de l'urine Le diagnostic radiologique fut vérifié par l'autopsie dans tous ses détails.

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AUS DEM BIOPHYSISCHEN INSTITUT DER UNIVERSITÄT ZU KOPENHAGEN.
CHEF: PROFESSOR DR PHIL. H. M. HANSEN

BETRACHTUNGEN, DURCH OSZILLOGRAPHISCHE MESSUNGEN GESTÜTZT, ÜBER APPARATEN FÜR DIE RÖNTGENDIAGNOSTIK[1]

von

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(In der Sitzung d. *Dän. Radiol. Gesellsch.* 14—12—38 vorgelegt.)

Jeder Röntgenarzt, der durch eigene Erfahrung oder durch Vertiefung in die Literatur, die geschichtliche Entwicklung der Röntgendiagnostik kennt, versteht die Bedeutung, die die Technik für die diagnostischen Leistungsmöglichkeiten hat. Er versteht, dass die technische Entwicklung sich stets im Vorrücken befindet und immer kompliziertere Maschinen hervorbringt. Früher war es dem Arzte recht leicht, sich mit den verhältnismässig einfachen Apparatkonstruktionen vertraut zu machen, so dass er sie selbständig beurteilen konnte, heute sehen wir, dass immer mehr Ärzte die modernen, komplizierten Konstruktionen als unerforschbare Geheimnisse ansehen, bei deren Beurteilung sie sich immer mehr von den Technikern und Physikern leiten lassen. Diese Tendenz der heutigen Entwicklung ist kaum eine glückliche, anstatt die Wirkungsweise seines Instruments zu verstehen, wird der Arzt Bediener einer Maschine werden, und diagnostische Möglichkeiten, die vielleicht ausnützbar sein würden, werden mit der steigenden Unkenntnis der physikalischen Prozesse bei der Diagnostik nach und nach der Vergessenheit überlassen.

Wünscht man, wie es gewissermassen hier der Zweck ist, sich einen Überblick über die heutige technische Entwicklung zu bilden, würde es natürlich sein, erst festzustellen, wie *das gute* Bild aussehen soll. Bekanntlich ist diese scheinbar einfache Voraussetzung in einem Kreis von Radiologen kaum zu verwirklichen, weil die persönlichen Ansichten so abweichend sind. Diese Abweichungen sind teils von zufälliger Art, so wie persönlichen Neigungen oder ästhetischen Wünschen — die als Vernunft-

[1] Bei der Red. am 1. VIII. 1939 eingegangen.

gründe nicht angesehen werden können —, teils von prinzipieller Art, um möglichst weich zu arbeiten (wie CHANTRAINE und seine Schule es tut) oder um hart »durchzuarbeiten«.

Sehen wir von den prinzipiellen Streitigkeiten weg, können die meisten Auffassungen sich wohl darin einigen, dass dasjenige Bild das beste ist, das in der möglichst deutlichen Weise die meisten Einzelheiten hervortreten lässt. — Die Erreichung dieser Idealforderung wird durch verschiedene Faktoren erschwert, unter denen wir die Aufmerksamkeit auf einige wenigen, wichtigeren lenken wollen.

Auf dem Wege der Röntgenstrahlen durch den abzubildenden Körper entstehen Fragen strahlungsphysikalischer Natur. Mit der chemischen Zusammensetzung der Objektelemente übereinstimmend werden die Strahlen bei dem Durchgang mehr oder weniger geschwächt; die Schwächung tritt ein, teils, weil Teile der Primärstrahlung absorbiert, teils, weil Teile gestreut werden. Es leuchtet ein, dass die Absorption der Primärstrahlung für die Entstehung des Bildes günstig ist, während die Verkleinerung der Primärstrahlung durch Streuung zwar zu der Entstehung des Bildes beiträgt, gleichzeitig indessen die diffuse Strahlung über das ganze Bild liefert, die als Sekundärstrahlung abflachend auf das hervorgebrachte *Strahlungsrelief* einwirkt; mittels geeigneter Bucky-Blenden kann man ja einen gewissen Prozentsatz von der Sekundärstrahlung wieder entfernen, wodurch wir auch die Bildentstehung, die durch Streuung der Strahlen geschieht, besser ausnützen.

Es ist allgemein bekannt, dass wir die Absorption der Strahlen in den einzelnen Objektelementen durch Änderung der Aufnahmespannung beeinflussen können. Leichtatomige Stoffe zeigen sich nun Änderungen der Strahlungsqualität gegenüber *weniger* beeinflussbar als schweratomige; die ersteren behaupten ihre schattenbildende (d. h. absorbierende und — namentlich — streuende) Wirkung bei höheren Spannungen verhältnismässig gut, die letzteren — schweratomigen — werden mit steigender Spannung verhältnismässig durchsichtiger, obwohl sie bei niedrigeren Spannungen dichte Schatten leisten.

Für verschiedene, einfachere Stoffe können wir die Strahlenschwächung berechnen und dadurch ausfindig machen, welche Strahlungsintensitäten nach Schwächung durch diese Stoffe zu erwarten sind (vorläufig bei gegebener, konstanter Spannung und konstantem Strom). — Bei jeder, gegebener, konstanter Spannung löst die Elektronbeschiessung des Brennfleckes das sogenannte »Bremsspektrum« aus, das Röntgenstrahlen aller Wellenlängen bis zu einer gewissen, kürzesten Wellenlänge, λ_{min} oder λ_o, hinab enthält; die kürzeste Wellenlänge und die gegenseitige Intensität der Wellen sind für jede Spannung charakteristisch. λ_o wird nach dem DUANE-HUNT'schen Gesetz, $\lambda_o = \frac{12.35}{kV}$, in *Å*-Einheiten, die relativ

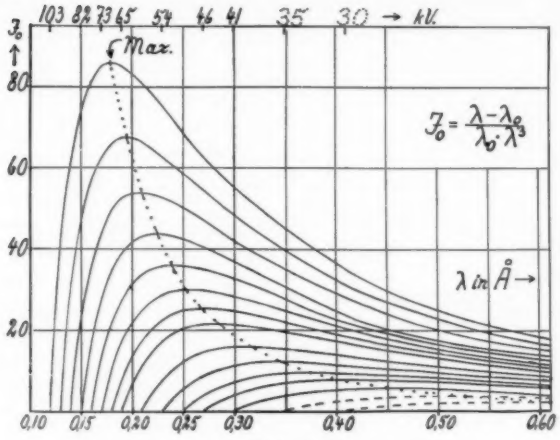

Abb. 1.

Intensitätsverteilung im Bremsspektrum gemäss KULENKAMPFF nach seiner Formel $I_o = \frac{\lambda - \lambda_o}{\lambda_o \cdot \lambda^3}$ berechnet. In der Formel bedeutet I_o die ungefilterte Intensität der gesuchten Wellenlänge λ eines Bremsspektrums, in welchem λ_o die kürzeste Wellenlänge ist. Werden die Ausrechnungen für verschiedene Spannungen ausgeführt, entstehen Kurven, deren Form in Abb. 1 zu sehen ist; die Wellenlänge in *Å* (die ja wieder einer gegebenen Spannung entspricht) ist als Abszisse, die relative Intensität der einzelnen Wellenlängen ist als Ordinate benutzt. Ob zwar die Strahlung durch eine konstante Spannung hervorgebracht ist, handelt es sich ja um keine »gleichartige« — noch monokromatische Strahlung; konstant ist bei einer gegebenen Spannung (bzw. kürzesten Wellenlänge) nur *die Zusammensetzung* der Strahlung. — Wünscht man indessen ausfindig zu machen, welche Kontraste durch einige bekannte Stoffe, wie Paraffin oder Wasser in verschiedenen Schichtdicken, hervorgebracht werden, muss man die allgemeine Schwächungsformel, $I_s = I_o \cdot e^{-\mu d}$, auf eine Reihe von Punkten in der Kurve eines Bremsspektrums anwenden. Beispielsweise machen wir eine solche Berechnung für k. k. Gleichspannung von 77 kV ($\lambda_{min} = 0.16$ *Å*), die Abb. 2 stellt bildlich die Ergebnisse dar; in diesem Beispiel stellt I_o (die punktierte Kurve) die relative Intensitätsverteilung des Bremspektrums bei 77 kV dar; Kurve 1 zeigt aber die Intensität desselben Spektrums nach Filterung durch 2 mm. Al (man sieht, dass diese Verminderung recht bedeutend ist). Da die Oberfläche des betreffenden Stoffes von einer Strahlung nach Kurve 1 zusammengesetzt getroffen wird, ist es ja diese Strahlung, von der wir in der Praxis ausgehen, und ich

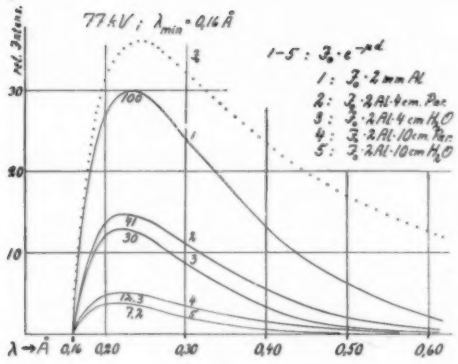

Abb. 2.

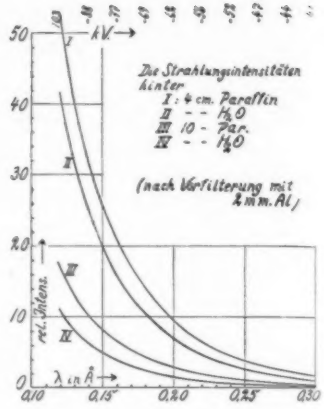

Abb. 3.

habe daher ihre ganze Intensität mit der Zahl 100 bezeichnet (die Zahl ist nur relativ, mit der Fläche unter der Kurve proportional). Wird diese Strahlung nun durch eine 4 cm. dicke Schicht von Paraffin geschwächt, nimmt die Intensität die von Kurve 2 angegebenen Werte an, und sie ist auf weniger als die Hälfte verkleinert worden, nämlich von 100 auf 41. Hätten wir statt durch Paraffin die Strahlung nach Kurve 1 durch 4 cm. Wasser abgeschwächt, wäre die Schwächung grösser geworden, — Kurve 3 — von 100 auf 30. Schichtdicken von 10 cm. Paraffin oder 10 cm. Wasser schwächen die ursprüngliche Intensität (Kurve 1) auf 12.3 bzw. 7.2 (die Kurven 4 und 5). Wären diese verschiedenen Schichten neben einander angebracht gewesen, würden Intensitätsunterschiede der Strahlung, d. h. ein Strahlungsrelief mit den Werten 100, 41, 30 u. s. w., entstanden sein. Auf einem Durchleuchtungsschirm, der die Intensitäten proportional wiedergibt, hätten wir sie unmittelbar beobachten können, auf dem Film treten sie mit neuen Werten hervor, weil die Film-Folie-Kombination die Unterschiede des Strahlungsreliefs *verstärkt*.

Wenn man entsprechende Berechnungen, wie im obigen Beispiel gezeigt, für eine Reihe von anderen Gleichspannungen, höher und niedriger als die in dem Beispiel benutzte Spannung von 77 kV, ausführt, wird es dadurch möglich auszudrücken, wie die Kontrastverhältnisse zwischen den hier besprochenen Schichten von Paraffin und Wasser sich anderen, durch andere Gleichspannungen hervorgebrachten, Strahlungen gegenüber verhalten. Die Abb. 3 stellt in Kurvenform die Ergebnisse solcher, umständlichen Ausrechnungen dar; auf der Abszisse ist die λ_{min} abgesetzt, als Ordinate dienen die Werte der Flächen unterhalb Kurven, die für andere Spannungen aber sonst ganz wie in der vorigen Abb. 2 berechnet un

ezeichnet sind (die Ordinatenwerte entsprechen also hier den Zahlen, ie in der Abb. 2 an den Kurven angebracht waren, ihre Flächengrössen ngebend). — Mit steigender Spannung sieht man in Abb. 3, dass der Kon- ast zwischen den Schichten vermindert wird (siehe z. B. die Kurven 3 nd 4 für 10 cm. Paraffin und Wasser: bei 0.16 *Å* (69 kV) ist das Verhält- s 4 : 2, bei 0.12 *Å* (103 kV) etwa wie 3 : 2). Ausserdem sieht man, dass e Strahlenausbeute mit der steigenden Spannung bedeutend anwächst.

Es ist in der Radiologie ein weit verbreitetes Missverständnis aus die- m Verhalten mit dem abnehmenden Kontrast bei höheren Spannungen n Schluss zu ziehen, dass die Aufnahmen vorzugsweise bei niedrigeren pannungen stattfinden müssen, u. a. weil die Streustrahlung unverhält- smässig stark mit der Spannung steigen solle. Es ist meine bestimmte — auf ausführlichen Versuchen fussende — Überzeugung, dass diese nnahme unrichtig ist; ich möchte bei dieser Gelegenheit die Einzelheiten, ie in einer anderen, bald erscheinenden Arbeit[1] niedergelegt sind, nicht äher besprechen, sondern nur hervorheben, dass die Streustrahlung in- erhalb des Diagnostikbereiches jedenfalls nur im beschränkten Mass mit der Spannung steigt (möglicherweise ist sie sogar konstant) und nur bei äusserst niedrigen, in der Praxis kaum verwendbaren Spannungen von etwa 40 *kV*, vielleicht ein wenig kleiner ist. Ich sehe dies Verhalten für die Röntgendiagnostik als sehr bedeutsam an, denn es bedeutet, dass die frühere, fast panische Angst vor den hohen Spannungen durch Fehldeu- tungen von Beobachtungen und Erfahrungen zu erklären ist. Diese An- sicht bedeutet nicht, dass ich die vorhandene Streustrahlungsmenge, die je nach Schichtdicke, Feldgrösse und Gewebeart der Primärstrahlung gegenüber gross sein kann, ungern möglichst vermindert sehe, sondern eine Folge meiner Ansicht ist, dass wir uns sehr wohl erlauben können, die grössere Strahlenausbeute der höheren Spannung auszunutzen. Der Ver- lust an Kontrastreichtum, der dadurch entstehen wird, lässt sich durch die Benutzung eines Films mit steilerer Gradationskurve leicht ausgleichen — oder man bringt in anderer Weise dem Film eine steilere Gradation bei. Natürlich wird die Entwicklung nach diesen Richtlinien *ausserordentlich genau* arbeitende Röntgenapparate fordern, aber solche liegen schon vor, wie es späterhin erörtert werden soll, und wir dürfen hoffentlich annehmen, dass die Filmproduzenten uns künftig mit Filmen bereichern werden, die mit steigender Empfindlichkeit und steilerer Gradation uns eine Ausnutzung der Apparate ermöglichen werden, die die heutige Leistungsmöglichkeit weit übersteigen wird. — Jeder, der den Vortrag des Dr. Saugman's[2] anhörte und seine Vorführung der blaugetönten Filme, die

[1] Helge Christensen: Beitr. z. Verständnis d. Leistungsfähigkeit d. med. Durchleuchtungsverfahrens. Kopenhagen 1939, Dissert.

[2] A. Saugman in Verh. d. Dän. Radiolog. Gesellsch. 9. 12. 36, Hosp. tid. 1936.
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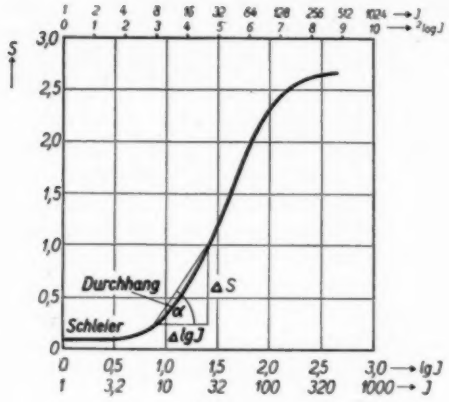

Abb. 4.

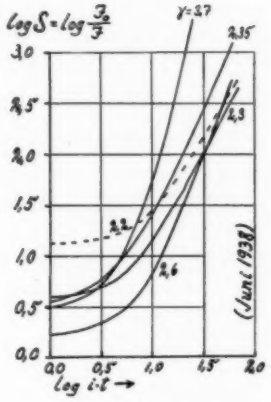

Abb. 5.

beim gelben Na-Licht beobachtet wurden, sah, muss eine eindrucksvolle Erinnerung davon haben, dass eine Herabsetzung der Kontraste schon binnen sehr weiten Grenzen ausgleichbar, bzw. überkorrigierbar ist.

Wenn das Missverständnis, dass die Entstehung eines flauen Bildes bei hoher Aufnahmespannung durch die hierbei überhandnehmende Streuung zu erklären ist, sich fortwährend bewährt hat, rührt es wohl zum Teil aus einer fehlenden Vertrautheit mit der Entstehungsweise des Filmbildes her. — Auf dem Durchleuchtungsschirm können wir das Strahlungsrelief, das beim Durchgang der Strahlen durch den untersuchten Körper gebildet ist, beobachten, denn der Schirm gibt die verschiedenen Intensitäten sozusagen tonrichtig wieder; d. h., ist die Intensität hier doppelt so gross wie da, leuchtet der Schirm hier doppelt so stark wie da. — Wird aber die Kombination von Verstärkungsschirm und Film von dem Strahlungsrelief getroffen, werden die Intensitäten bekanntlich nach der (auch bedeutungsvollen) Entwicklung des Filmes in keiner einfachen Weise wiedergegeben. Die Schwärzung des Films ist mit der Intensität der Strahlen nicht proportional, sondern sie findet gemäss einer Kurve statt, deren prinzipielle Form in der Abb. 4 zu sehen ist. Wenn die Intensität klein ist, wird nicht geschwärzt, steigt sie, nimmt die Schwärzung[1] erst langsam, nach und nach schneller — und schliesslich konstant mit der Intensität zu. Unmittelbar sieht man nun, dass, falls ein Bild, z. B. wegen Aufnahme mit weichen Strahlen, über grosse Intensitätsunterschiede verfügt, beispielsweise ab log I = 1.0 bis log I = 1.5 (numerisch 10 bis 32), Schwärzungsunterschiede ab 0.3 bis etwa 1.1 mittels

[1] »Schwärzung 1« = Schwächung des durchfallenden Lichts auf 1/10, »S. 2« auf 1/100 u. s. w.

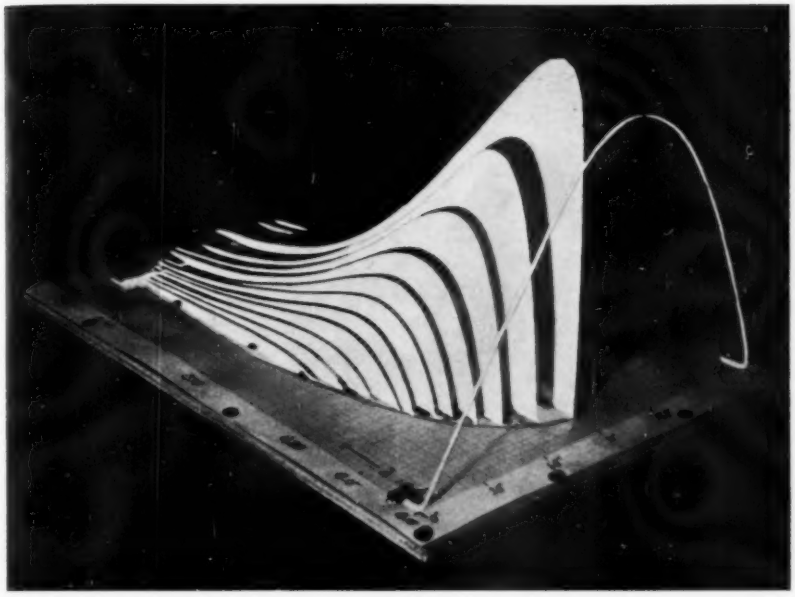

Abb. 6.

der Kurve abgelesen werden können. Sind die Kontraste im Strahlungsrelief aber nur kleiner, z. B. wegen Aufnahme mit harten Strahlen, so dass log I nur zwischen 1.2 und 1.5 variiert (in reinen Zahlen ab 16 bis 32, d. h. etwa 1: 2) lesen wir durch die Kurve der Abb. 4 Schwärzungen von 0.8 bis 1.0 ab. Das Bild ist somit viel kontrastärmer geworden — *wegen der Wirkungsweise, die durch die Kurve zum Ausdruck kommt, und n i c h t wegen der Streuung!* Eine steilere Gradationskurve könnte das erste (und grössere) Kontrastverhältnis auch im zweiten Falle, bei der harten Strahlung, wiederherstellen.

Schon zu der Zeit sehen wir, dass erhebliche Unterschiede an den Gradationskurven verschiedener Filme bestehen; die Abb. 5 stellt die Ergebnisse einiger im Frühling 1938 von uns vorgenommenen Bestimmungen der Kurven von 5 handelsüblichen Filmsmarken dar; die Betrachtung dieser Kurven lehrt uns sofort, dass es schon möglich — und vorteilhaft — ist, Filme mit geeigneten Kurven für bestimmte Zwecke zu verwenden. Leider fehlen teilweise noch zwei Voraussetzungen für diese Massnahme, die künftig als selbstverständlich angesehen werden muss: 1) Jede Filmfabrik stellt gewöhnlich nur einen Filmtypus her (aus technischen Gründen vielleicht unvermeidlich), dessen Schwärzungskurve und sonstige

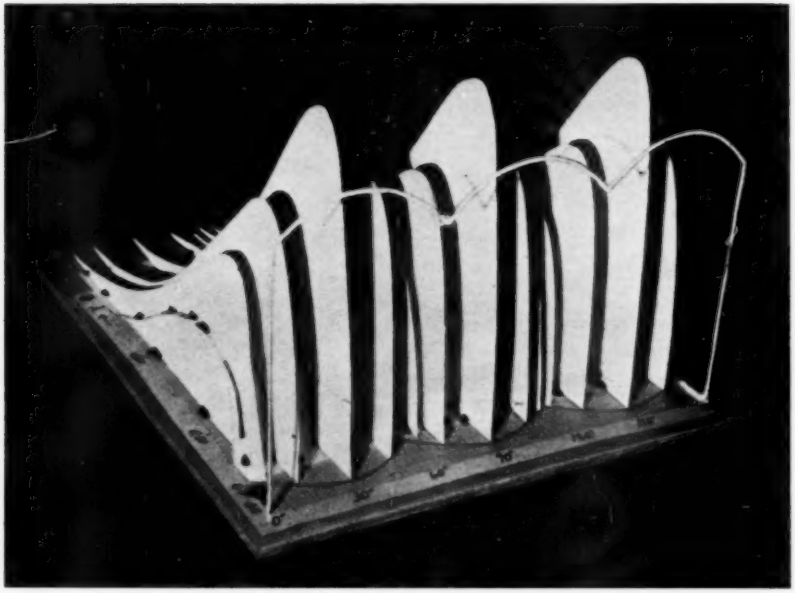

Abb. 7.

Eigenschaften allerdings vom Verkäufer stark gelobt — aber sonst sorgfältig geheimgehalten werden! Natürlich können wir selbst die Eigenschaften ausmessen — und das wird immer notwendiger werden —, aber *warum soll diese Ware, der Film, anonym verkauft werden?* Die Konkurrenten messen ja augenblicklich den neuen Film aus, die Radiologen sind ja gar nicht immer auf derlei Messungen eingestellt — und sind dann ganz im Stiche gelassen. 2) Um den Unterschied zwischen zwei verschiedenen Filmen ausnutzen zu können müssen die Apparate genauer als die Mehrheit der heutigen Apparate arbeiten können, die elektrische Leistung, die der Röhre zugeführt wird, muss peinlich genau wiederholbar und nach beliebigen Zeiträumen wiederherstellbar sein. Diese Forderung wird mit zunehmender Empfindlichkeit und Steilheit der Filme noch mehr geschärft werden, und um diese Verbesserungen fruchtbar machen zu können, müssen die Apparatkonstrukteure verstehen, dass dadurch sehr grosse Ansprüche an ihre Erzeugnisse gestellt werden.

Wie oben besprochen ist es durch umständliche, aber jedoch recht überschauliche Ausrechnungen möglich, bei einer gegebenen Spannung das sich ergebende Kontrastverhältnis zwischen zwei Stoffen bekannter Art (z. B. Wasser und Paraffin u. s. w.) ausfindig zu machen. Da die Röntg-

genmaschine ja keine konstante Spannung liefert, ist es unmittelbar einleuchtend, dass ausser der Intensität auch die spektrale Zusammensetzung der erzeugten Röntgenstrahlung genau mit den momentanen Werten der Spannung gesetzmässig variieren muss. Denken wir uns, die Spannung steigt und fällt, genau und wiederholt mit einer gegebenen Kurve übereinstimmend (wie es in der Praxis oftmals der Fall ist), dann würden wir von Moment zu Moment die Kurvenreihe der Abb. 1 (mit allen denkbaren Zwischenstufen) bis zum Höchstwert durchlaufen, und der abfallende Zweig der Spannungskurve würde uns dieselben Kurven der Intensität und spektralen Verteilung in umgekehrter Ordnung wiedergeben. Um den verwickelten Vorgang abzubilden muss man sich eines dreidimensionalen Koordinatensystems benutzen. Dies scheint und klingt so schwierig, aber wenn man nur ein plastisches Bild von diesem System herstellt, wird der ganze Vorgang — wie aus Abb. 6 ersichtlich — sofort vereinfacht.[1] Diese Abbildung illustriert die Verläufe der spektralen Verteilungen und dazu entsprechenden Intensitäten während eines sinusförmigen Spannungsstosses (wie sie theoretisch von Halbwellen- und 4-Ventilapparaten geliefert werden). Vorn sieht man die sinusförmige Spannungskurve, die mit 100 kV gipfelt, und für verschiedene Momentanwerte der Spannung sind Kurven der spektralen Intensitäten (wie in Abb. 1) aus Karton ausgeschnitten und nach der Tiefe hin aufgestellt. Denken wir uns die Ränder der Kartonfiguren durch einen Mantel umhüllt, würde eine sanft abgerundete, stromlinienartige Bildung entstehen, deren Gesamtvolumen ein Ausdruck für die beim Stromstoss erzeugte Strahlungsmenge und Art wäre; (wenn die Exposition aus einer Reihe von solchen Spannungsstössen besteht, wird nicht die Qualität geändert, nur die Strahlenmenge wächst mit der Anzahl der Stösse). Hätten wir nun zwei Modelle nach dem Prinzip der Abb. 6 hergestellt, von denen z. B. das erste aus Kurven aufgebaut sei, die die ursprünglichen Intensitäten nach Filterung durch 2 Al und Schwächung durch 4 cm. Paraffin darstellen (ähnlich wie es in der Abb. 2 nur für eine einzelne Spannung gemacht wurde), und das zweite Modell aus entsprechenden Kurven mit 2 Al aber 4 cm. Wasser gebaut wurde, dann würde das Verhältnis zwischen Volumen der beiden Bildungen ein Ausdruck für den Strahlungskontrast zwischen 4 cm. Paraffin und 4 cm. Wasser bei einer sinusförmigen Spannung (bei 100 kV) sein. Obwohl die Methode umständlich und zeitraubend ist, sieht man, dass es keineswegs unmöglich ist, rechnerisch einen Eindruck von den Vorgängen bei verschiedenen Spannungsverläufen auszuarbeiten. — Die Abb. 7 zeigt in ähnlicher Weise die Strahlenerzeugung bei der Spannungsform des Drehstromapparates in derselben Zeitspanne (1/100 sek.) wie die Abb. 6.

[1] Die Abb. 6 lässt nur die linke Hälfte des Modells hervortreten.

Schon der unmittelbare Vergleich zeigt recht deutlich, dass die Strahlenausbeute — *ceteris paribus* — in derselben Zeiteinheit beim Drehstromapparat bedeutend grösser ist als beim 4-Ventilapparat, und gleichzeitig sieht man, dass die spektrale Zusammensetzung eine andere — und für dieselbe Spitzenspannung härtere ist. Baut man — ähnlich wie beim einfacheren Beispiel des sinusförmigen Stromstosses — auch Modelle nach Art der Abb. 7 aus Kurven auf, die die Intensitäten nach 2 Al Vorfilterung und Durchgang durch 4 cm. Paraffin bzw. Wasser darstellen, könnte man wiederum das Strahlen-Kontrastverhältnis zwischen diesen beiden Medien sowohl bei gleichgerichtenem Drehstrom, wie bei sinusförmigem Stromstoss aus den Rauminhalten der Modelle ableiten. — Einfacher, aber weniger übersichtlich lassen diese Berechnungen sich durch Kurven abbilden. In der Abb. 8 ist der sinusförmige Spannungsverlauf (bis 100 kV_{sp}) durch die Kurve I angegeben. Die Kurve II stellt nun (in willkürlichem Masstabe) für jeden Momentanwert der Spannung die dazu entsprechende Gesamtheit aller spektralen Intensitäten nach Passage durch 2 Al und 4 cm. Paraffin dar. Man sieht, dass weit die grösste Strahlenmenge im Laufe höchstens $^1/_3$ der Periode abgegeben wird. — (Zur Deutung der Abb. 8 vergleiche man die Abb. 6.)

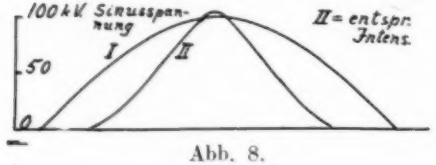

Abb. 8.

Es ist nun unter den Radiologen eine weit verbreitete Annahme, es sei für die *Qualität* des Bildes vorteilhaft, dass die Spannung eines Röntgenapparats während der Aufnahme möglichst konstant verlaufe; so zu verstehen, dass die Stromkurve des Drehstromapparates der des 4-Ventilapparats, und die reine, unbewegliche Gleichspannung der Kurve des Drehstromapparates überlegen seien, *auch* wenn es sich um den Bildcharakter handelt. Dies trifft aber nicht zu, in so fern als man, wie .u a. von Taylor and Singer[1] gezeigt wurde, eine Gleichspannung und eine dazu gehörende Absorptionskurve finden kann, die mit genügender Annäherung dem Strahlengemisch entspricht, das durch irgendeine, pulsierende Spannung entsteht. Schon der Umstand, dass eine Gleichspannung (vgl. Abb. 1) ein ausgesprochenes Strahlengemisch hervorbringt, dürfte den Verdacht erregen, dass die Bedeutung der Gleichspannung in dieser Beziehung nur gering sein kann. Mittels der für diese Arbeit errechneten, zahlreichen Kurven, ist es innerhalb engerer Grenzen möglich, ein einfaches Beispiel hierfür zu zeigen: Man denke sich, die Spannung sinke während einer Aufnahme geradlinig z. B. von 82 auf 73 kV herab (λ_{min} 0.16 bis 0.18 Å). Zeichnen wir für die Anfangs und Endspannung

[1] Lauriston S. Taylor and George Singer, Radiology *22*, 445, 1935. Siehe auch Kurt Bischoff, Röntgenpraxis *7*, Hft. 1, 1935.

n gewöhnlicher Weise die Intensitätskurven für z. B. 2 Al und 4 cm. araffin auf (Abb. 9), wissen wir, das während der Aufnahme nach und ach eine Reihe von Intensitätskurven zwischen den beiden Endkurven elegen hat; als Ausdruck hierfür können wir mit grosser Annäherung[1] ine Kurve zeichnen, die der resultierenden spektralen Zusammensetung während unserer Aufnahme ntspricht, und sie wird aus den urchschnittswerten der Anngs- und Endkurven gebildet nd ist in der Abb. 9 punktiert ngegeben. Wird aber nun auch ie entsprechende Kurve für 7 kV Gleichspannung (annähherungsweise die Mittelspannung wischen 82 und 73 kV!) im Bilde ingeführt — die ausgezogene Kurve der Abb. 9 — sieht man, dass diese weitgehend mit der Durchschnittskurve zusammenfällt. Rücksichtlich Qualität werden somit die beiden Bilder, 82→73 kV, 77 kV Gleichsp., ganz einander gleich sein. — — —

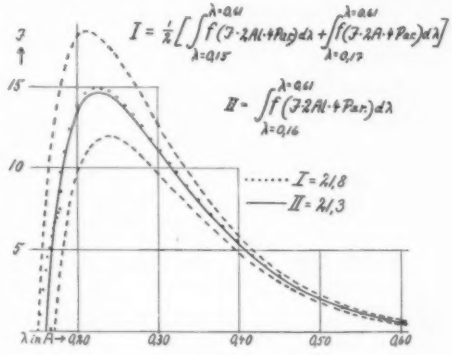

Abb. 9.

Wenn man sich nun die besprochenen Tatsachen erinnert, die Streuung ändere sich mit der Spannung des Diagnostikbereiches nicht merkbar, der Bildcharakter lasse sich durch die Gradationskurve eben so wohl wie durch die benutzte Spannung beeinflussen, die Strahlenausbeute nehme mit der Spannung schnell zu, und die Spannungskurve eines Apparats sei für den sich ergebenden Strahlungskontrast weitgehend gleichgültig, wird es sofort ersichtlich, wir können teils künftige Ansprüche an die Apparate andeuten, teils die Apparattypen von heute in diesem Lichte betrachten.

Die letzte Aufgabe ist vornehmlich heute die Aufgabe und muss ja die Voraussetzung für künftige Forderungen bilden. Während wir bisher die Fragen theoretischer angesehen, mit wirklich sinusförmigen Spannungskurven gerechnet und ausserdem die *zeitlichen* Verhältnisse ganz ausser Acht gelassen haben, müssen wir nun die tatsächlichen Formen der Spannungskurven sowie ihre zeitlichen Verläufe und Wiederholbarkeit näher ansehen. Bekanntlich ist es immer schwierig gewesen, geeignete Schaltorgane für die schnelle Schaltung der bedeutenden Stromzufuhr der Apparate zu bauen. Da die Leistungen moderner Apparate riesig sein können, darf man sagen, dass es wohl kaum möglich und jedenfalls

[1] Die Abnahme der Intensität findet ja nicht proportional mit der Spannung statt, im kleinen Bereich ist diese Abweichung aber nicht gross, und möglich würde es sein, sie genau auszurechnen.

bisher nicht in ganz befriedigender Weise gelungen ist, mechanische Schütze herzustellen. Die Bewegung des mechanischen Teils des Schützes muss eine kurze Minimalzeit dauern, unterhalb welcher der Vorgang sich nicht abspielen kann. Zeiten länger als diese »untere Grenzzeit« werden durch eine Uhr oder durch ein mAs-Relais gesteuert. Fehlermöglichkeiten liegen nun teils bei der reinen Wiederholung dieser Bewegungen vor (sei es durch die Uhr oder den Schalter selbst), teils kann das vielleicht genau bewegte Schütz durch eine von Fall zu Fall sich ändernde Funkenbildung kompromitiert werden; leider nimmt diese mit der Apparatleistung zu und macht sich ausserdem besonders bei den Kurzzeitaufnahmen geltend, weil bei diesen selbst eine kurzdauernde Funkenbildung prozentisch eine sehr bedeutende Verlängerung der beabsichtigten Beleuchtung auswirkt. Daher entstehen die selbst bei modernen Apparaten nicht unbekannten Schwierigkeiten zwei Aufnahmen nach einander genau gleichartig aufzunehmen. —

Oft wird ja nun gefragt, ob da diese beständige Forderung, grosse Leistungen während extrem kurzen Zeiten, sich wirklich mit der Tatsache einigen lasse, dass die Röntgenbilder ja bei weitem nicht dieselbe Schärfe besitzen, wie die Bilder, die wir mit einem ganz billigen — nur 10 Kronen teuren! — photographischen Apparat erreichen können! Die Unschärfe eines solchen Bildes braucht 0.1 mm. nicht zu übersteigen, beträgt manchmal nur 0.05 mm., während aber die besten herzustellenden Röntgenbildern eine Konturunschärfe von wenigstens 0.2 oder eher 0.4 mm. besitzen, wenn sie mit Folien aufgenommen sind. Die Frage ist natürlich unrichtig gestellt, denn während die folienbedingte Unschärfe (U_f) und die geometrische Unschärfe (U_g) bei den gegebenen Aufnahmebedingungen gewissermassen konstant sind, kann die Bewegungsunschärfe (U_b) ganz andere Grössenordnungen annehmen.[1] STUMF gibt ja schon für das Herz Lateralbewegungen bis 100 mm./Sek. an, wobei noch bei einer wahren Belichtungszeit von 0.01 — eine noch sehr seltene Aufnahmezeit — eine theoretische Unschärfe[2] von 1.0 mm. in der Herzumgebung entstehen kann. Endlich darf man nicht vergessen, dass die Bewegungen, die die Patienten entweder durch Unverständigkeit oder mitgenommen durch ihre Krankheit ausführen, gross sein können — und nur durch Kurzzeitaufnahmen (und d. h. wiederum Riesenleistungen) zu begegnen sind.

Als Begründung für Kurzzeitaufnahmen bleibt noch die Theorie von der *Superposition*, wie sie durch Dr. FRANKE aufgestellt ist, übrig. Wenn

[1] Die früher oft gesehene Betrachtungen, die um die Voraussetzung, $U_f = U_g = U_b$, kreisten, sind daher besser für rechnerische Erwägungen als für praktische Tatsachen geeignet.

[2] »theoretisch« weil ich begründete Vermutungen davon habe, dass die tatsächliche U_g im Bilde deutlich kleiner scheint als der berechnete Wert; nähere Untersuchungen hierüber sind im Gange.

man sich vorstellt, dass jedes kleine Bildelement z. B. in der feinen Lungenzeichnung zu Voraussetzung hat, dass in der Dauer der Aufnahme eine Reihe von kleinen Einzelheiten in der Lunge auf einmal auf einer Linie liegen müssen, wird es ohne weiter einleuchtend, dass die Aufnahmezeit so kurz wie möglich sein muss, weil sonst die sich bewegenden und schwingenden Teilchen aus der Lage auf der Linie gleiten können; das Superpositionsbild der Teilchen wird sich entweder vergrössen oder ganz verwischen. Hierdurch erklärt sich die von vielen beobachtete Erscheinung, dass mit kürzeren Zeiten um je mehr Einzelheiten im Lungenbilde auftauchen.

Auch wenn man die zeitlichen Leistungen der Apparate studieren will, ist die Oszillographie die Methode der Wahl, und hoffentlich wird es bald selbstverständlich sein, dass jedem Apparat beim Verkauf eine Reihe von individuellen Oszillogrammen beigelegt wird, so wie es zu fordern ist, dass jeder Film von seiner Gradationskurve und jede diagnostische Röntgenröhre von einer individuellen Brennfleckphotographie begleitet sind! Ein vorzügliches Mittel für derartige Studien besitzt man in den Elektronenstrahl-Oszillographen, die sich mit der schnellen Vervollkommung der Kathodenstrahlenröhren leicht für diese Zwecke einrichten lassen. Ehe wir Beispiele der ausgeführten Messungen an verschiedenen Apparattypen anführen, dürfte es angebracht sein diese Messmethode, die Oszillographie (»Schwingungsschreibung«) kurz zu beschreiben, da sie noch kaum den weniger physikalisch geschulten Lesern bekannt sein kann.

Die Oszillographie der Physik lässt sich historisch in zwei Abschnitte teilen, Oszillographie *ohne* (d. h. *vor* der Entdeckung) und *mit* (d. h. *nach* der Entdeckung) Kathodenstrahlen. Die ältere Oszillographie beschäftigte sich beispielsweise mit Stimmgabelschwingungen, Druckschwingungen in Flüssigkeiten und Gasen, elektrischen Schwingungen und Impulsen (Reaktionsströmen und Elektrocardiographie). Bei allen diesen Aufgaben waren die beiden Hauptfehler immer dieselben: Die Apparate führten eine — oftmals unbekannte — Verzerrung der Messergebnisse ein, weil sie die mechanischen Schwingungen dämpften, und die Apparaten vermochten keinen hochfrequenten Schwingungen zu folgen. Die Fehler beruhen beide auf der mechanischen Trägheit der Registriersysteme der Instrumente.

Diese Mängel der früheren oszillographischen Untersuchung wurden vollständig beseitigt, da die Physik und die Technik die sogenannten Kathodenstrahlenröhren hervorbrachten, mit deren Hilfe jede denkbare oszillographische Untersuchung ausgeführt werden kann, besonders wenn andere, physikalische Hilfsapparate, so wie Mikrophone bei akustischen, Photozellen bei optischen und piezoelektrische Aufstellungen bei mechanischen Schwingungen, mittels geeigneter Verstärker angekoppelt werden.

Die Abb. 10 zeigt schematisch eine moderne Kathodenstrahlenröhr für Oszillographie. Die durch den Glühstrom erhitzte Kathode ist vo einem Wehneltzylinder umgeben (der eine negative Spannung von 5 % des Nennwertes der Anodenspannun erhält). Durch die Unterbringun geeigneter Platten mit steigende positiven Spannungen (in unsre Röhre wahlweise bis 1250 bzw. 250 V.) werden die Elektronen mit grosse Geschwindigkeit als ein schmale Bündel gegen die Endwand der Röhr geschleudert. Da die Wand mit Kalziumwolframat (o. ä.) überzogen ist wird ein leuchtender Fleck an der Stelle gebildet, wo die Elektronen an prallen. Im Hals der Röhre passiert das Elektronenbündel zwischen zwe Plattenpaaren, von denen das eine Paar im Verhältnis zum anderen um 90° gedreht steht. Wird nun einer Platte eine positive oder negative Span nung zugeführt, wird das Bündel angezogen, bzw. abgestossen, und der Leuchtfleck bewegt sich entsprechend. Die Abbeugung der Elektronen ist mit der Spannung proportional, und da sie weiter mit grösster Schnelligkeit und vollkommen trägheitslos stattfindet, versteht man, dass hiermit das ideale Instrument für Schwingungsuntersuchungen geschaffen ist. Das eine Plattenpaar bewirkt die Bewegungen auf und ab, das andere die Bewegungen hin und her, senkrecht auf den ersten. Für viele Zwecke genügen Ausschläge nach einer Richtung, wobei man entweder mit dem rotierenden Spiegel oder aber durch photographische Übertragung auf dem konstant vorwärtzgeführten Filmstreifen die Spannungsschwankungen in Kurvenform beobachtet.

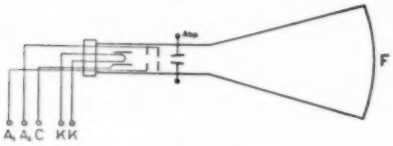

Abb. 10.

Die von uns verwendete Röhre ist von M. v. ARDENNE gebaut, ihre Spannungsempfindlichkeit beträgt 0.50 oder 0.25 mm/Volt bei 1250 bzw. 2500 V. Anodenspannung. Einleitend wurde im *Biophysischen Institut der Universität zu Kopenhagen* die anzuwendende Messaufstellung durchgearbeitet und geeicht. Da eine Ablenkplatte unserer Röhre für Ausschläge von 5—7 cm. eine Spannung von 2—300 V. benötigt, musste man, um die Hochspannungen der Röntgenapparaten messen zu können, einen guten, induktionsfreien Hochohmswiderstand als Spannungsteiler zur Verfügung haben, der ausserdem leicht transportabel sein musste. Die Fabrik »*Vitrohm*« in Kopenhagen stellte uns 4 Widerstände, je etwa 25 Megohm, her, die in dünnen Porzellanrohren, etwa $^1/_2$ m. lang, eingebettet waren. Im *Biophys. Inst.* wurden sie mit einem amerikanischen Präzisionswiderstand (»*Taylor High Resistor*«) verglichen und sie haben sich sowohl genau wie genügend belastbar gezeigt. Die Abb. 11 zeigt die Schaltung bei einer Spannungsmessung zwischen Plus und Erde. In anderen Fällen muss der Mittelpunkt des Widerstandes geerdet, und

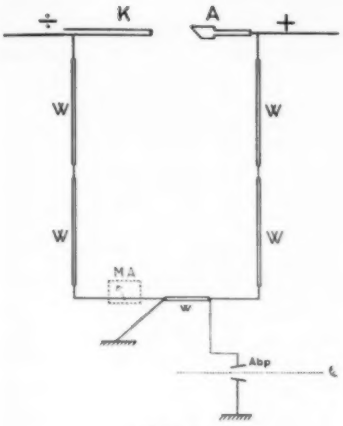

Abb. 11.

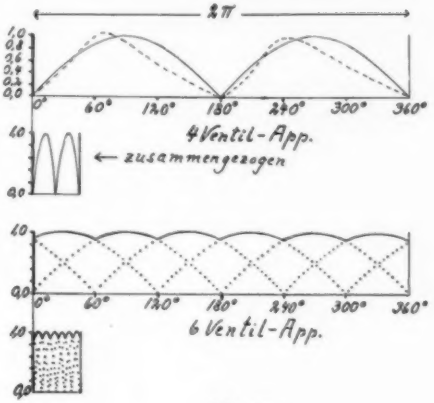

Abb. 12.

Punkte in der Umgebung hiervon mit den Ablenkplatten verbunden werden, damit Schwingungen sowohl des positiven wie des negativen Poles aufgezeichnet werden können; überhaupt muss die Schaltung ja immer mit Rücksicht auf die Apparatschaltung aufgestellt werden.

Um auch Strommessungen ausführen zu können haben wir teils einen zweistufigen Wechselstromverstärker, teils einen einstufigen Gleichstromverstärker, beide genügend aperiodisch, gebaut. Verstärkung ist notwendig, weil man gewöhnlich in den modernen Röntgenapparaten den gleichgerichteten Anodenstrom, der das mA-Meter passiert, oder der Wechselstrom eben ausserhalb dieses Gleichrichters, nur durch kleine Fallwiderstände leiten darf.

Die photographische Registrierung wurde mit einem alten *Zeiss-Ikon* Kinoaufnahmeapp., aus welchem der Mitbringer entfernt war, so dass der Film etwa 70 cm./Sek. konstanter Fahrt geführt wurde, vorgenommen. Die Optik war ziemlich lichtstark, etwa 1 : 1.8, Brennweite 12.5 cm. — An Stelle des Kinofilms wurde gewöhnlich das Papier für Siemens Elektrokardiograph benutzt, das etwa dieselbe Empfindlichkeit und ausserdem angenehme, im voraus einkopierte Linien besitzt — und dazu billiger ist.

Zum Verständnis der Oszillogramme ist nur hervorzuheben, dass, falls man sinusförmige Kurven mathematisch genau abbilden sollte, die Abzisse für eine ganze Periode etwa 6 mal (genau 2π) länger als die Ordinatenwerte sein müsse, damit die Kurve die bekannte langwellige Form erhalten könne. Wenn aber der Papierstreifen zu langsam läuft, werden die Kurven zusammengeschoben wie es die Abb. 12 sowohl für den reinen sinusförmigen Stromstoss wie für den gleichgerichteten Drehstrom zeigt.

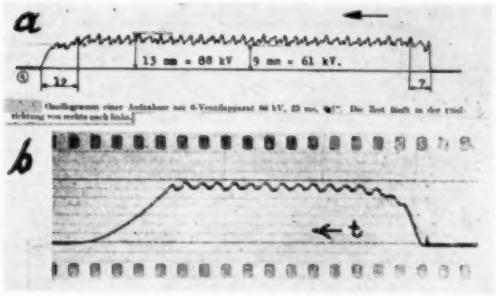

Abb. 13.

So lange die Einzelheiten der Kurven noch abgelesen werden können, ist die Zusammenschiebung bedeutungslos. Hätten wir genaue Kurven zeichnen wollen, hätte der Papierstreifen etwa 10 mal schneller laufen müssen, — was technisch unmöglich und praktisch überflüssig war.

Ausserhalb der Fabriken sind oszillographische Messungen an Röntgenapparaten nur verhältnismässig selten ausgeführt worden. LAURITZEN[1] hat im Jahre 1928 Oszillogramme der Entladungen seiner ersten Hochspannungsröntgenröhren veröffentlicht, und mit Rücksicht auf röntgendiagnostischen Zwecken liegt im Jahre 1936 von CHANTRAINE[2] die erste Mitteilung vor, in welcher er u. a. die Spannungskurve, die in Abb. 13 a dargestellt ist, wiedergibt. Auf diese Kurve hinweisend, kritisierte er die von der Drehstrommaschine geleistete Spannungskurve und forderte die Hersteller auf, Kondensatoren an den Drehstrommaschinen anzubringen, um eine weitere Ausglattung der Spannung herbeizuführen. Die Abb. 13 b, die einer Broschüre der Firma Koch und Sterzel entnommen ist, zeigt die hierbei erreichte Verbesserung, indem diese Firma eine solche Konstruktion hervorbrachte. Man sieht, dass eine kurze Zeit vergeht, bis die Spannung ihren Höchstwert erreicht, weil die ersten Energiemengen in den Kondensatoren aufgespeichert werden, und umgekehrt wird nach der Unterbrechung der Exposition eine entsprechende Energiemenge abgeliefert. Da die Spannung während dieser Erscheinungen grösstenteils zu niedrig ist, um Schwärzung hervorbringen zu können, sind sie somit bedeutungslos.

Um die unsrigen Oszillogramme richtig abzulesen, muss die Bemerkung vorgeschickt werden, dass die Null-Linie *oben* liegt, positive Spannungen liefern Ausschläge nach unten; die Oszillogramme fangen immer links an und schreiten nach rechts vor.

Als erstes Beispiel zeigt die Abb. 14 oben den Oszillogramm einer Durchleuchtung mit einem 4-Ventilapparat bei 60 kV und nur 1 mA; wegen des Vorhandenseins der Hochspannungskabel, die eine gewisse Kapazität und daher eine ausglattende Wirkung ähnlich wie im obigen Beispiel haben, finden wir, dass es sich um eine leicht pulsierende Gleich-

[1] CHARLES G. LAURITZEN, Physic. Review, 1928.
[2] CHANTRAINE, Fortschr. Rtg.strahlen, *53*, 585, 1936.

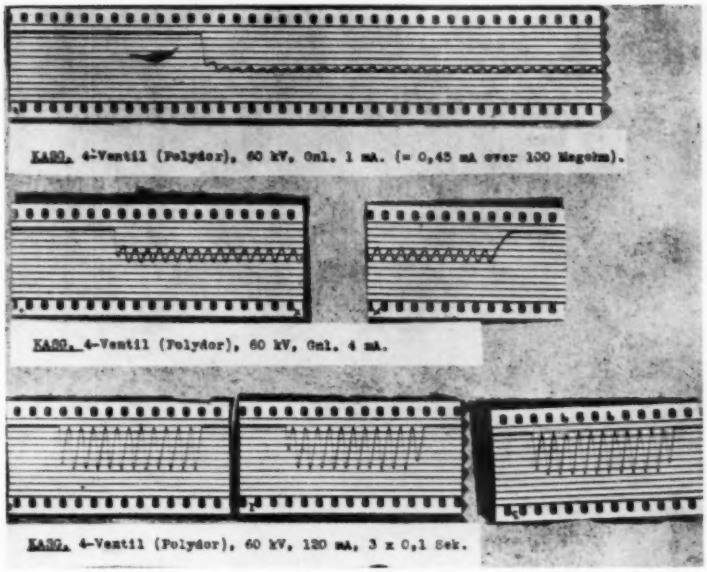
KASO. 4-Ventil (Polydor), 60 kV, 0nl. 1 mA. (= 0,45 mA over 100 Megohm).
KASO. 4-Ventil (Polydor), 60 kV, 0nl. 4 mA.
KASO. 4-Ventil (Polydor), 60 kV, 120 mA, 3 x 0,1 Sek.

Abb. 14.

spannung handelt. Steigt die Stromstärke auf 4 mA finden wir, dass die ausglattende Wirkung der Kabelkapazität schon weniger ausreicht, und bei 3 Aufnahmen mit 120 mA — unten in der Abb. 14 — je von »1/10« Sek. finden wir ihre Wirkung ganz aufgehoben (die Kabel waren übrigens in diesem Falle recht kurz, insgesammt etwa 7 m.); wir zählen 12 Spannungsspitze, die besagen, dass die Aufnahmen nicht 0.10 sondern 0.12 Sek. gedauert haben.

Im Halbwellenapparat werden bekanntlich nur alle zwei Spannungsstösse gleichgerichtet und ausgenutzt; die Abb. 15 zeigt in ähnlicher Weise eine Durchleuchtung mit einem Halbwellenapparat, der keine isolierte Kabel besitzt, so dass keine ausglattende Wirkung besteht; bei zwei verschiedenen Spitzspannungen finden wir nach verschiedenen einleitenden Unregelmässigkeiten die für diesen Apparattypus charakteristischen Spannungskurven rechts beim Schluss der wiedergegebenen Oszillogrammen. Um eine Ausglattung in diesen Fällen zu erhalten müsste die ausglattende Kapazität natürlich grösser sein um dieselbe Wirkung zu haben.

In modernen Installationen wird es oftmals der Fall sein, dass hochspannungsisolierte Leitungen grösserer Längen — und somit grösserer Kapazitäten — verwendet werden. Die Oszillogramme zeigen dann, dass

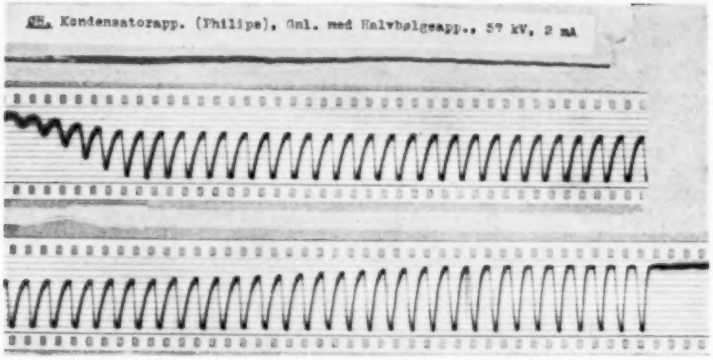

Abb. 15.

Durchleuchtungen sowohl bei 4- wie bei 6-Ventilapparaten praktisch bei einer Gleichspannung vor sich gehen (ich vermeide die Wiedergabe derlei selbstverständlicher Oszillogramme). Dies Verhalten dürfte insofern bedeutungsvoll sein, da die Röhrenfabrikanten gewöhnlich streng hervorheben, dass die Durchleuchtung bei Gleichspannungen die Röhren durch mögliche Stossjonisation besonders gefährde; *wir müssen somit feststellen, dass dieser bedrohliche Zustand leider oft vorhanden ist* und auf eine möglichst vorsichtige Durchleuchtung Anspruch macht, — sonst erklärt er vielleicht ein zu schnelles Zugrundegehen einer Röhre.

Mittels der Oszillographie können wir übrigens alle Vorgänge in einem Apparat verfolgen vom Anfang bis zum Schluss. In einer dänischen Stadt wird der normale, sinusformige Wechselstrom nach Gleichrichtung den Verbrauchern als »pulsierender Gleichstrom« zugeschickt. Die Abb. 16 zeigt oben diesen eigentümlichen Gleichstrom; nach Passage des Umformers entsteht auf neue ein gewissermassen korrekter Wechselstrom (Abb. 16, 2. Osz.), in welchem man jedoch überlagernde Schwingungen an beiden Schenkeln erkennen kann, die kaum vorteilhaft sein können. Nach mechanischer Gleichrichtung (es handelt sich um einen alten *Transverter*) entstehen — bei Durchleuchtung — die sehr engen Spannungsspitzen, die durch lange Pausen getrennt sind; eine deutlichere Illustration der geringen Leistungsmöglichkeiten der alten Apparate ist kaum möglich.

Die Arbeitsweise des Transformators lässt sich auch beurteilen, und die Abb. 17 liefert ein solches Beispiel. Erst ist die vom Netz erhaltene Spannung von 380 V. aufgezeichnet, sowohl ohne als mit Belastung durch den Apparat (durch eine Aufnahme). Die Kurvenform der Primärspannung wird durch die Belastung keineswegs beeinflusst. Danach wird der Oszillograph direkt an die Sekundärklemmen angelegt, und bei geringer

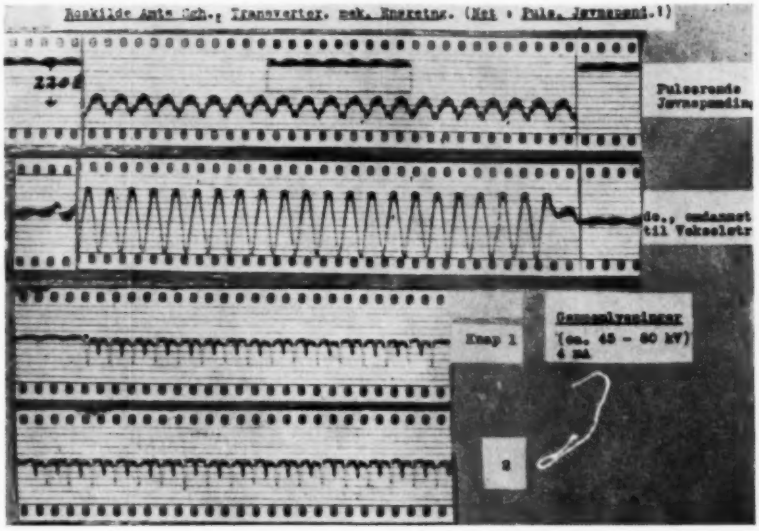

Abb. 16.

Belastung (Durchl. mit etwa 2 mA) findet man die primäre Kurvenform fast ungeändert wieder. Bei grösserer Belastung (Aufn. mit etwa 250 mA) sieht man, dass die Spannungskurve sich merkbar ändert und zugespitzt wird.

Um die Genauigkeit der kürzeren Aufnahmezeiten bei einem Apparat zu untersuchen ist es zweckmässig — wie in Abb. 18 —, eine Reihe von Oszillogrammen bei verschiedenen Zeiten aufzunehmen, um sie für den Vergleich unter einander aufzukleben. Man sieht an diesem Beispiel, wo es sich um einen älteren 4-Ventilapparat handelt, dass die kürzeren Zeiten, kleiner als 0.1 oder jedenfalls 0.05, prozentisch ziemlich ungenau und zufällig wiedergeben werden. Bei unsren bisherigen Messungen im Felde haben wir noch nur einen modernen 4-Ventilapparat gefunden, der genau und reproduzierbar bis auf 0.02 Sek. hinab arbeitet, sonst ist das abgebildete Beispiel der Regelfall.

Kehren wir uns nun den Drehstromapparaten zu, deren Zweckmässigkeit vielen der oben besprochenen Ansprüche an grosse Leistung und Genauigkeit gegenüber besonders interessant ist, finden wir, dass sie gewöhnlich zeitlich genau bis auf 0.03 oder 0.02 Sek. arbeiten können. Eine gewisse Ungenauigkeit rührt von Ungleichmässigkeit der Spannungskurve her; im Idealfall sollten wir pro Periode (1/50 Sek.) 6 sinusförmige Spannungsgipfel (siehe Abb. 12) erhalten. In Wirklichkeit sind die Gipfel ja

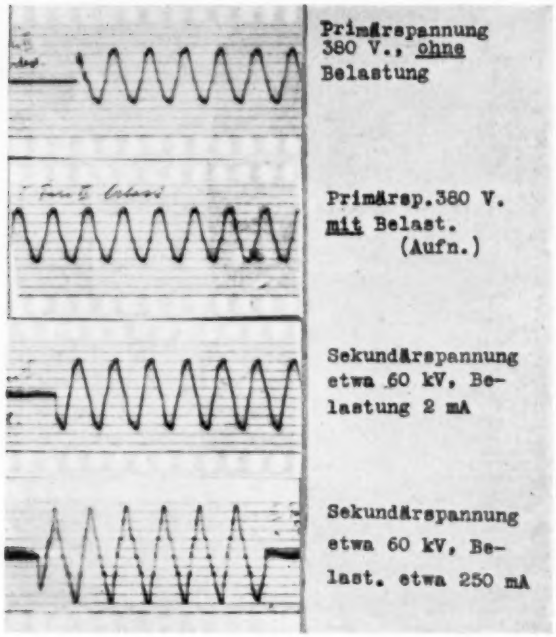

Abb. 17.

weder bei 4- noch bei 6-Ventilrohrapparat sinusförmig, sondern etwas zugespitzter (in Abb. 12 oben nach einer Messung gezeichnet), und ausserdem können sie kleinere Unterschiede in der Höhe, Spannungsunterschiede, zeigen. Da die erzeugte Strahlenmenge ja mit einer Potenz der Spannung variiert, wie oben erörtert, kann das kurzzeitig aufgenommene Bild verschieden beleuchtet werden, wenn auch der Apparat genau dieselbe Zeit eingeschaltet war: besteht eine Periode der Spannungskurve des Drehstromapparates beispielsweise (etwa wie in der Abb. 21) aus drei niedrigeren und drei ein wenig höheren Spannungsstössen, hat man die Möglichkeit bei einer Zeit von 0.03 Sek. (anderthalb Periode) 2 Gruppen mit höheren und eine mit niedrigeren Gipfeln zu erhalten, oder umgekehrt — und trotz derselben Einschaltzeit werden die Bilder ein wenig verschieden werden. Leichter ist das Verhalten ja beim 4-Ventilapparat einzusehen, wo ganze Stösse wegfallen oder verzerrt auftreten können. Nur die oszillographische Darstellung enthüllt diese Geheimnisse, und nur durch ihre Kontrolle kann man Versuche machen, die Unregelmässigkeiten zu beseitigen.

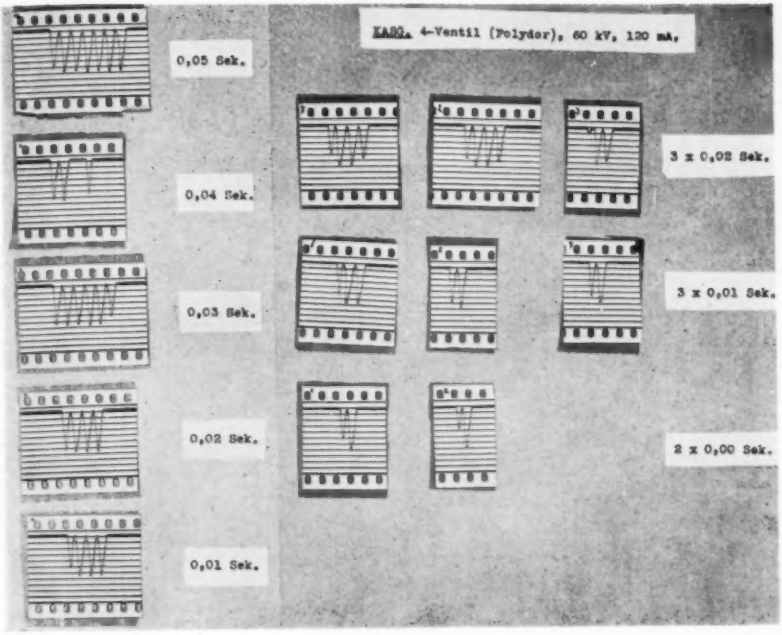

Abb. 18.

Die Abb. 19 zeigt Oszillogramme eines bis auf 0.02 Sek.hinab genau arbeitenden Drehstromapparates; zwar fangen die Kurven mit einer grossen Einschwingung an (werden oft auch mit einer Sonderschwingung abgeschlossen), die Kurvenform und die Dauer sind aber ohne Rücksicht auf die Belastung ganz genau. Etwa 3/4 km. lange Zuleitungen aus dem Transformatorturm sind wahrscheinlich durch Wanderwellen o. ä. für die Sonderschwingungen verantwortlich. Die eigentliche, gleichgerichtete Dreiphasenkurve des Apparats ist ja sonst sehr regelmässig und genau.

Die Abb. 20 stellen Oszillogramme eines anderen, ebenfalls schön arbeitenden Drehstromapparats dar, bei welchem die durch mAs-Relais gesteuerte Zeitregulierung bis auf etwas 0.03 Sek. genau wirkte. Die Durchleuchtung findet bei reiner Gleichspannung statt, die Kabelkapazität ist ausser an der Kurve auch in der Nachwirkung zu verspüren. 3 Aufnahmen, jede von 20 mAs, allerdings mit verschiedener Stromstärke, 100, 200 und 500 mA, zeigen dann und wann kleine Anfangsunregelmässigkeiten; wenn die Aufnahme mit 500 mA nicht 1/5, sondern eher etwas 1/4 Sek. der Zeit für die 100 mA-Aufnahme dauert, liegt nur eine nicht genaue

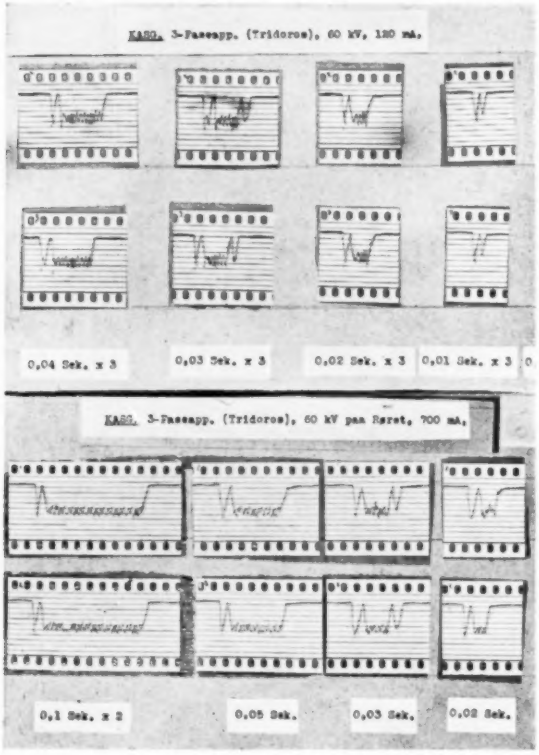
KASO. 3-Fasesapp. (Tridoros), 60 kV, 120 mA.
0,04 Sek. x 3
0,03 Sek. x 3
0,02 Sek. x 3
0,01 Sek. x 3
KASO. 3-Fasesapp. (Tridoros), 60 kV pas Rørst, 700 mA.
0,1 Sek. x 2
0,05 Sek.
0,03 Sek.
0,02 Sek.

Abb. 19.

Glühstromeinstellung vor; das ist ja eben der Vorteil des mAs-Relais', wir erhalten bis an die kritische Grenze (hier bis 0.03 Sek.) immer dasselbe Produkt aus Zeit mal Strom.

Schliesslich zeigt die Abb. 21 die schon oben erwähnte 3-Phasenkurve, in der wir in jeder Periode eine Gruppe von 3 ein wenig grösseren und 3 kleineren Gipfeln finden; sonst sind die Zeiten genau bis etwa 0.03 Sek. Die ohne die Oszillographie ganz verborgene Erscheinung liess sich leicht beseitigen, so dass eine schöne Kurve nach Art der Abb. 19 (ohne die Ein- und Ausschwingungen) entstand. —

Da unser Oszillograph ja nur *eine* Kathodenstrahlröhre besitzt, konnten wir nicht gleichzeitig Spannungs- und Stromkurven aufzeichnen; da wir indessen einen 4-Ventilapparat fanden, der bis auf 0.02 Sek. genau reproduzierbar arbeitete, zeigen wir in der Abb. 22 Beispiele einiger Mes-

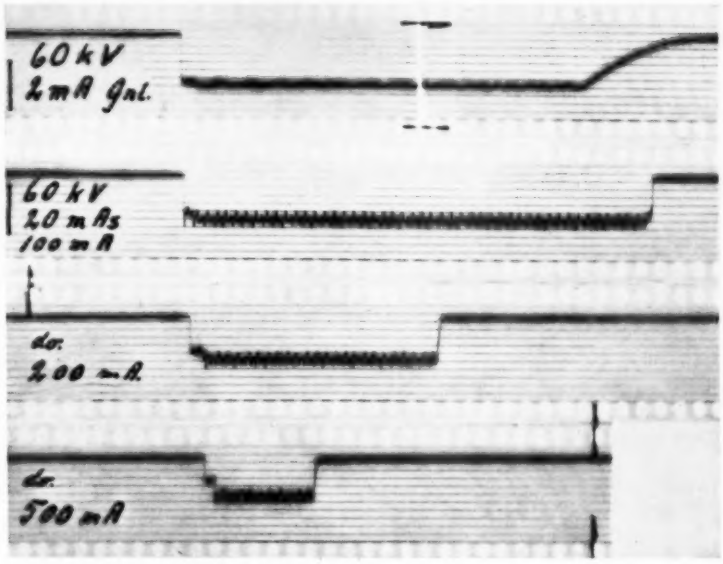

Abb. 20.

sungen, bei welchen wir erst die Spannungskurven und dann die Stromkurven unter den gleichen Bedingungen massen. In diesem Falle war es möglich den Wechselstrom beim geerdeten Mittelpunkt des Transformators über genügenden Fallwiderständen (deren Grössen neben den Oszillogrammen angeführt sind) zu leiten, um ausreichende Steuerspannungen ohne Verstärkung zu erhalten. Die »mA-Kurve« wird daher als eine Wechselstromkurve dargestellt. Wie die gewöhnlichen Spannungsoszillogramme es abbilden, sind die erste und zweite Halbperiode ein wenig unsymmetrisch, wir sehen, dass der Apparat indessen ganz genau im Verhältnis zur *Halbperiode* — sei es die erste oder die zweite — ein — und ausgeschaltet wird. Dasselbe sehen wir in den Strom-Oszillogrammen, deren Amplitude — wahrscheinlich wegen der Charakteristik der Röntgen- (und Ventil-?) Röhre — mit der Spannung deutlich, wenn auch nicht bedeutend, anwächst.

Durch die Multiplikation der Spannungs- und Stromkurven Punkt um Punkt ist die Kurve der abgegebenen Leistung abzuleiten. In der Praxis ist es leicht durchzuführen, indem man die Oszillogramme mit dem Projektionsapparat auf mm-Papier im grossen Massstabe aufzeichnet. In dieser Weise kann man auch zum näheren Studium die Oszillogramme im korrekten Abszissenmassstabe zeichnen (vergl. Abb. 12). Eine der-

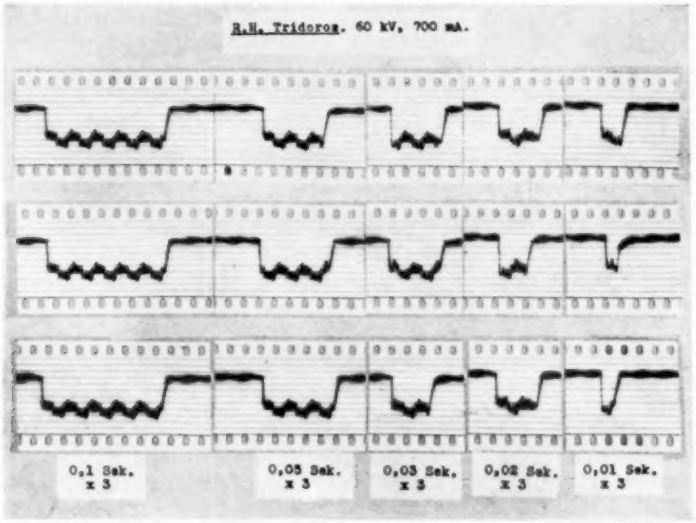

Abb. 21.

artige Analyse einiger der Messergebnisse der Abb. 22 ist in der Abb. 23 zu finden; die voll ausgezogene Linie stellt die Spannung, die gestippelte den Strom und die punktierte die ideale Sinusspannung dar. Die hoch emporsteigende Kurve gibt die Leistung (Watt) an und lässt uns deutlich ablesen, dass im 4-Ventilapparat weit die grösste Energiemenge innerhalb der Hälfte der Halbperiode abgeliefert wird[1]; dem gegenüber liegt ja eben der grosse Vorteil der Drehstromapparate, bei denen zwar wellig, jedoch in der Zeit viel gleichmässiger gearbeitet wird.

Mit besonderem Interesse muss man nun die Strommessung eines Super-D-Apparats vornehmen, denn dieser Apparat, der wohl besonders mit Hinblick auf die stillstehende und wärmespeichernde Anode gebaut wurde, ändert bekanntlich selbsttätig seine Stromstärke während der Aufnahme. Anfängliche mA-Werte fallen auf etwa die Hälfte oder noch weniger ab. Da man um dem Fall zu folgen etwa 1 Sek. belichten muss, ist eine ablesbare Wiedergabe eines ganzen Oszillogramms im Druck nicht möglich, die Abb. 24 gibt daher einige Ausschnitte eines solchen Oszillogramms wieder. (Die kleinen Zacken an der Kurve beruhen auf nebensächlichen Beeinflussungen des Oszillographens durch Streufelder im Schalttisch.) Die allmähliche Abnahme der Stromstärke tritt in der Abbildung ablesbar hervor, etwa 0.4 bis etwa 0.6 Sek. dauernd. Die

[1] Man vergleiche dazu die Abb. 8 und die daran geknüpfte Erörterung.

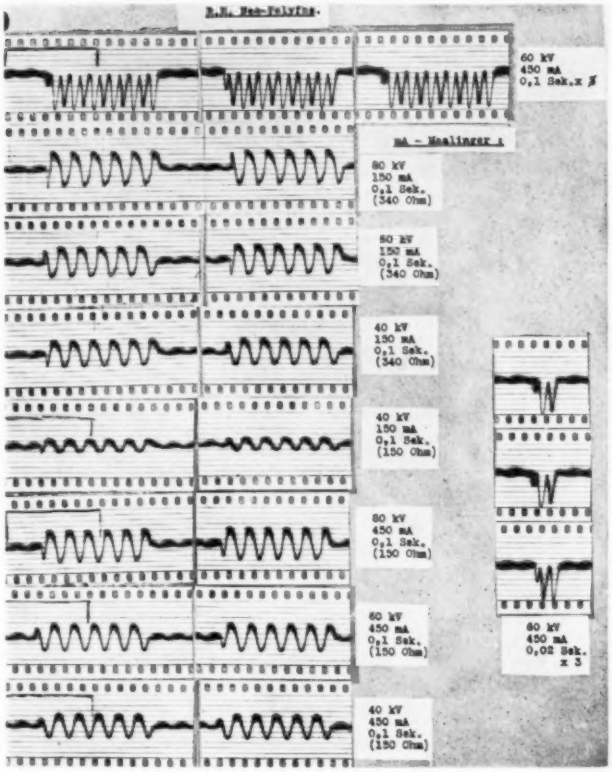

Abb. 22.

Spannungskurve zeigt sich dabei ganz unbeeinflusst. (Dieser Apparat arbeitete nicht mit einer stillstehenden Röhre sondern mit einer Drehanode.)

Als letztes Beispiel finden wir in der Abb. 25 Oszillogramme, die die Arbeitsweise eines Kondensatorapparats erklärt. — Bekanntlich ist es das Prinzip eines Kondensatorapparats, dass die für die Aufnahme zu verwendende elektrische Leistung schon im voraus durch Aufladung eines Kondensators bereit gehalten wird.

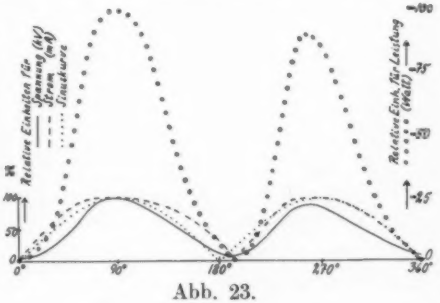

Abb. 23.

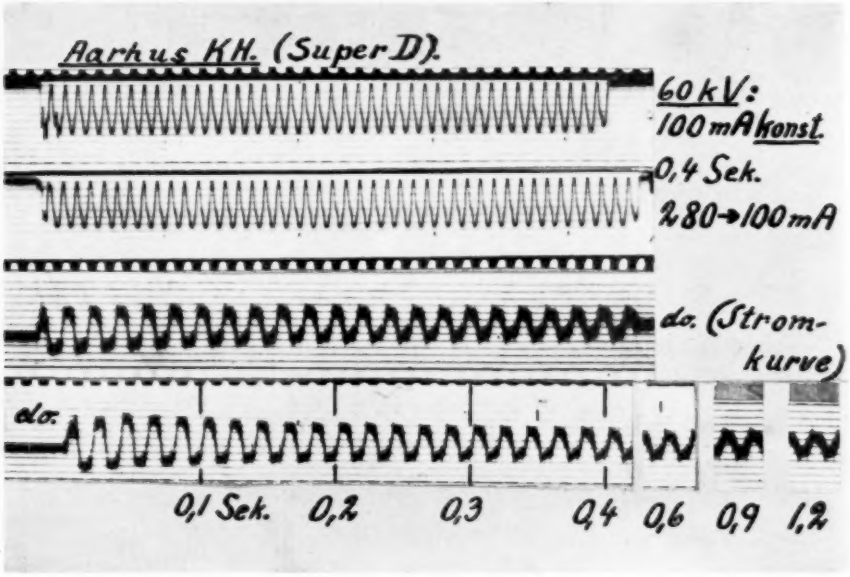

Abb. 24.

Je nach Kondensatorgrösse, Schaltmassnahmen und Belastbarkeit der Röhre kan man die aufgespeicherte elektrische Energiemenge ohne Rücksicht auf den Transformator oder das Netz verwenden. Die Oszillogramme der Abb. 25 stammen von einem Apparat, der besonders für Lungenaufnahmen gebaut ist. Der Kondensator wird zuerst auf die gewünschte Anfangsspannungsstufe aufgeladet, während der Aufnahme sinkt die Spannung dann, je nach Stromstärke schneller oder langsamer. Wahrscheinlich wegen der Röhrencharakteristik sinkt die Spannung nicht geradlinig, sondern mit sinkender Spannung allmählich langsamer. Aus dem Verlauf der Spannungskurve kann man, da es sich um eine Kondensatorentladung handelt, den Schluss ziehen, dass der Strom durch die Röhre im Anfang grösser ist und langsam ein wenig abfällt. Die beiden Ursachen, die anfänglich höhere Spannung und der grössere Strom, bewirken also eine im Anfang der Aufnahme grössere Schwärzung, die verhältnismässig schneller zurückgeht. Zwei Aufnahmezeiten sind möglich, indem die Entladung des Kondensators in geschickter, mechanischer Weise unterbrochen werden kann, oder man kann die ganze aufgesammelte elektrische Ladung abfeuern. Die Unterbrechung geschieht auf etwa 6/100 Sek. (die kleine Welligke

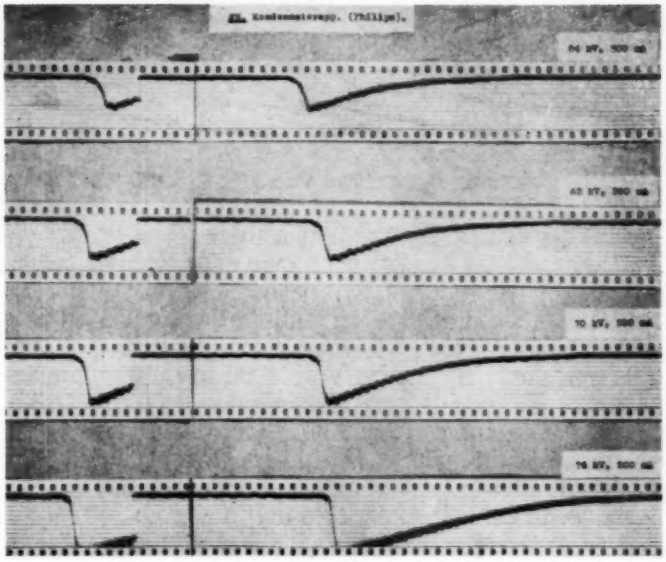

Abb. 25.

der Kurve rührt von dem Glühstrom her, 2 Spitzen entsprechen somit der Periode) und ist ausserordentlich genau. Aus der Arbeitsweise des Apparats geht es hervor, dass die längere Aufnahmezeit — also ohne Unterbrechung — vorzugsweise bei den höheren Anfangsspannung Bedeutung hat, denn sonst ist die Spannung so viel abgefallen, dass der zusätzliche Schwanz der Kurve zur Schwärzung keinen nennenswerten Beitrag liefern kann. —

Ehe wir die letzten neuen Schöpfungen auf dem Gebiete der Apparatkonstruktion, die Grosskondensatorapparate, besprechen, dürfte es vielleicht zweckmässig sein, an die hiermit so eng verbundenen gewöhnlichsten physikalischen Grössen zu erinnern. Fliesst in *1 Sek.* die Einheit der Elektrizitätsmenge *1 Coulomb* durch einen Leiter, hat der Strom die Stärke von *1 Ampère.* Die Einheit für *Kapazität* (das Vermögen Elektrizitätsmengen aufzuspeichern) ist *1 Farad;* wird einem Kondensator von dieser Kapazität die Elektrizitätsmenge 1 Coulomb zugeführt, wird er auf die Spannung *1 Volt* aufgeladen; wäre seine Kapazität kleiner, würde die Spannung höher geworden sein. Die Einheit *1 Farad* ist nun eine sehr grosse und oft unpraktische, man rechnet viel öfter mit der 1 Million mal kleineren Grösse *Mikrofarad* (= μ F), die jedem Rundfunkbastler bekannt ist. Führte man dem Kondensator von 1 μ F die Elektrizitätsmenge *1 Cou-*

lomb zu, würde er auf eine Spannung von 1 Million Volt aufgeladen werden, wäre die Menge nur 0.1 Coulomb, würde die Aufladespannung nur 100 000 Volt betragen u. s. w. — Entladen wir den auf 100 000 Volt aufgeladenen Kondensator von der Grösse 1 μ F, können wir wieder die Elektrizitätsmenge 0.1 Coulomb herausleiten, und dauert die Entladung 1 Sek., ist die Stromstärke 0.1 Amp. in 1 Sek., d. h. 100 mAs. Ist die Kapazität des Kondensators um so und so viel mal grösser als 1 μ F, braucht er, um auf irgendeine Spannung aufgeladet zu werden, eine um so und so viel mal grössere Elektrizitätszufuhr als der Kondensator von 1 μ F, bei der Entladung leistet er aber auch eine entsprechend grössere mAs-Menge.

Während der Entladung fällt die Spannung des Kondensators genau im Verhältnis zur abgeflossenen Elektrizitätsmenge, und d. h., wenn der Entladungsstrom konstant ist, im Verhältnis zur abgenommenen mAs. Der auf 100 000 V. aufgeladene Kondensator von der Grösse 1 μ F besitzt somit nach Entnahme von 25 mAs noch eine Spannung von 75 kV u. s. w. — Die drei Hauptgrössen, Spannung, Kapazität und mAs werden durch die folgende Beziehung verbunden, die uns erlaubt, die eine Grösse zu berechnen, wenn die beiden anderen bekannt sind:

$$\text{Spannung (in kV)} = \frac{\text{mAs}}{\mu\,\text{F}}.$$

Wie früher erwähnt haben die modernen, hochspannungsisolierten Leitungen eine gewisse Kapazität, die doch noch im Verhältnis zur Grösse 1 μ F klein ist. Für solche kleine Kapazitäten werden daher vorzugsweise eine andere, kleinere Einheit benutzt, nämlich *1 cm.;* 1 μ F = $9 \cdot 10^5$ cm. Die Kapazität der Kabel sind gewöhnlich der Grössenordnung 180—200 cm. pro m., d. h., 50 m. Kabel besitzt nur noch eine Kapazität von etwa 1 % von 1 μ F. Für die bei Aufnahmen benutzten grösseren Elektrizitätsmengen ist diese Grösse daher ohne Bedeutung, bei den kleinen Stromstärken der Durchleuchtung verspürt man aber ihre Wirkung deutlich, wie die Oszillogramme es zeigten.

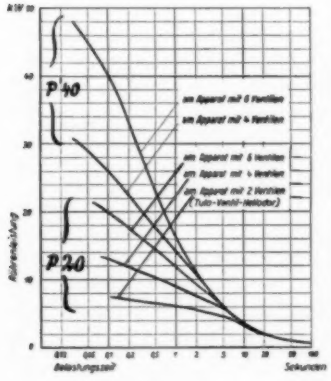

Abb. 26.

Die Hauptmerkmale der neuen Hochleistungskondensatorapparate sind auf verschiedenen Gebieten zu suchen; die Kondensatoren sind so gross dimensioniert, dass eine grosse Anzahl von üblichen Aufnahmen nur die Verwendung eines gewissen Bruchteils der im Kondensator aufgespreicherten Elektrizitätsmenge fordert,

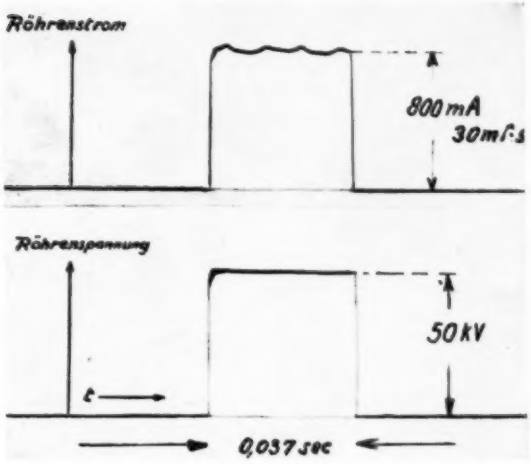

Abb. 27.

und diese können daher unter sehr genau definierbaren elektrischen Umständen stattfinden. Von grösster Bedeutung ist weiter die Entfernung aller mechanischen Bestandteile aus den Schaltvorgängen, diese werden mittels gittergesteuerten Röhren verschiedener Art ausgeführt, wodurch jede Trägheit praktisch aufgehoben ist, so dass die Ein- und Ausschaltung blitzschnell geschieht. Durch Elektronenröhren werden weiter sowohl die Aufladespannungen wie die Aufnahmezeiten durch mAs-Zählung gesteuert, so dass diese Prozesse weit genauer als in den Transformatorapparaten ausgeführt werden. Reichen für die schwereren Aufnahmen die Leistung der Kondensatoraufladung nicht aus, kann man schliesslich mit dem Aufladungstransformator arbeiten und dabei den grossen Kondensator als Ausglattungskapazität verwenden; in dieser Weise kan man beliebig lang bei 100 mA oder mehr bei praktisch reiner Gleichspannung belichten.

Diese neue Vervollkommung der Grossdiagnostikapparate dürfen wir zweifellos als die Einleitung einer neuen Epoche für die Röntgendiagnostik begrüssen, denn sie ermöglicht nach meiner Ansicht einen grossen Teil der Wünsche, die als Schlussfolgerungen der ersten Hälfte dieser Arbeit ausgesprochen wurden. Wenn auch der

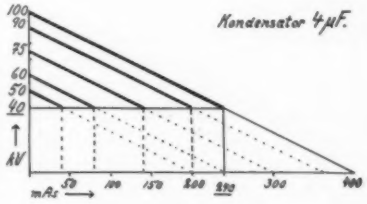

Abb. 28.

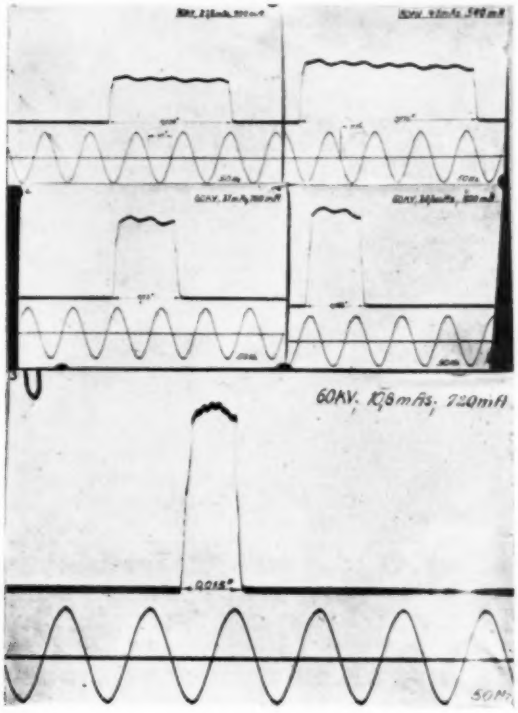

Abb. 29.

Kritiker bemerken kann, dass die schwereren Aufnahmen nicht mit der Kondensatoraufladung allein durchgeführt werden können, darf man indessen verschiedene Betrachtungen hervorheben. Die Arbeit mit der Kondensatorentladung ist besonders günstig bei kurzen Zeiten, in denen Stromstösse fast beliebiger Grössen geleistet werden können, die Grenze wird hier von den Röhren gesetzt. Die schwereren Aufnahmen, die nun vorläufig eine gewisse Zeit dauern müssen, können, falls die Kondensatorladung nicht ausreicht, mit Gleichspannung und wenigstens 100 mA ausgeführt werden, was gar keine schlechte Bedingung ist. Wichtiger dürfte es sein, dass die genaue Arbeitsweise uns erlaubt, steilere Filme zu benutzen und in dieser Weise die weit grössere Strahlenausbeute der höheren Spannung auszunutzen. Dabei darf nicht vergessen werden, dass jede Steigerung der Filmsempfindlichkeit auch eine damit proportionale Vergrösserung der Leistungsfähigkeit des Apparats bedeutet.

Endlich bietet die Kondensatorentladung wahrscheinlich der Drehanode noch die günstigste Belastungsweise. Die Abb. 26 rührt von einer zufälligen Broschüre über Drehanoden her und zeigt sehr deutlich die Bedeutung der elektrischen Belastungsform für die Belastbarkeit. Wenn man sich der Kurvenformen der 2-, 4- und 6-Ventilapparate erinnert, die beispielsweise in den Abb. 15, 18 und 19 abgebildet waren, wird es ohne weiters verständlich, dass je gleichmässiger die Energie verteilt ist, je besser wird der Anodenteller ausgenutzt. Bisher habe ich keine Angaben über die Belastbarkeit der Drehanoden beim Kondensatorbetrieb gesehen, aber wir dürfen sicher noch mit einer kleinen Steigerung rechnen.

Vielleicht werden wir auf diesem Gebiete noch einer Frage begegnen, die schwer zu beurteilen ist. Die Drehanoden rotieren gewöhnlich etwa 2 700 mal je Minute, d. h. eine Umdrehung dauert etwa 0.022 Sek. Die Kondensatorapparate ermöglichen aber noch kürzere Zeiten, die zweifellos künftig für verschiedene Zwecke häufig benutzt werden; d. h. aber, dass je kürzer die benutzte Zeit der kritischen Zeit von 0.022 Sek. gegenüber ist, je kleiner muss wiederum die Belastung gemacht werden, weil ja sonst nur Bruchteile der Anodenperipherie belegt werden. Eine Verkleinerung der Belastung bei Zeiten kürzer als 0.022 ist aber gar nicht der Zweck der Erschaffung der Kondensatorapparate gewesen. Wie bedeutend die Frage ist und wie sie zu lösen ist (selsbttätige Verdoppelung der Drehgeschwindigkeit bei Ultrakurzzeiten?) wird hoffentlich bald von den Konstrukteuren der Röhren erörtert werden.

Vorläufig haben wir in Dänemark noch keine Grosskondensatorapparate, und wir sind daher leider nicht in der Lage, originale Oszillogramme vorzuführen. Zur Zeit werden bekanntlich solche Apparate von den Firmen PHILIPS und SIEMENS hervorgebracht. Insofern die verhältnismässig kurzgefassten, konstruktiven Mitteilungen zu beurteilen sind, haben die beiden Firmen die Aufgabe in ausserordentlich verschiedenartiger Weise gelöst; ausser der prinzipiellen Verwendung von einem Kondensator und von Elektronenröhren nebst einer gewissen Übereinstimmung rücksichtlich der äusseren Arbeitsweise, die den oben angegebenen Forderungen genügen, ist es interessant festzustellen, dass die Konstruktionen und die innere Arbeitsweise der Apparate sonst weitgehend verschieden sind. Der *Maximus D-Apparat* der PHILIPSfabriken ist mir nur durch die kurze Mitteilungen der Literatur und die entsprechende Broschüre bekannt; mittels einer verwickelten Anordnung vermag der Apparat die Kondensatorladung unter Gleichspannung der Röhre darzubieten, wie die Abb. 27, nach der Broschüre wiedergegeben, es darstellt; vorläufig (Dezember 38!) liegen keine Erläuterungen über die Leistungsgrössen des Apparats im Kondensatorbetrieb vor. — Die von SIEMENS entwickelte Konstruktion, *Kodiaphos*, enthält einen Kondensator von 4 μ F, der bis auf 100 kV aufgeladen werden kann. Man kann sich hier-

durch einen einfachen Überblick über die erreichbaren Leistungen bei jeder Anfangsspannung verschaffen, wenn man — wie in Abb. 28 — ein Schema aufzeichnet, das die möglichen mAs ab eine gegebene Anfangsspannung bis etwa 40 kV, die als untere Grenze für nennenswerte Schwärzung gesetzt wird, angibt. — Wie früher betont, vermögen die beiden Apparate, wenn die Kondensatorleistung nicht ausreicht, praktisch reine Gleichspannung bei etwa 100 mA beliebig lang zu leisten.

Die Firma SIEMENS in Kopenhagen hat uns freundlich einige Strom-Oszillogramme aus praktischen Messungen im Felde überlassen, — und wir sprechen sofort unseren besten Dank für das Interesse aus. Die Oszillogramme sind in der Abb. 29 gesammelt und zeigen die blitzartig schnellen Ein- und Ausschaltungen, bis auf kurze Zeiten hinab; die Welligkeit stammt wie gewöhnlich in diesen Messungen aus den periodenabhängigen Schwankungen der Temperatur des Glühfadens, die Sinuskurve unten dient als Masstab für die Zeit.

Als Ergebnis der einleitenden Erwägungen und angeführten Messbeispiele dürfen wir festhalten, dass für einfachere, diagnostische Aufgaben, bei denen *verhältnismässig lange Zeiten* zur Verwendung kommen, 4-Ventilapparate ausreichend sein können. Wenn es sich um *Kleinleistungen* handelt, können weiter die Halbwellenapparate bekanntlich brauchbar sein. — Müssen *grössere Belastungen* und *Kurzzeitaufnahmen* (und solche sind im Hospitalsbetrieb nie zu vermeiden) benutzt werden, sind die Drehstromapparate unbedingt vorzuziehen, teils wegen des Netzes, teils wegen der grösseren Strahlenausbeute bei derselben kW-Leistung und teils wegen der günstigeren Belastungsweise der Drehanoden. Den Drehstromapparaten gleichwertig — oder eher überlegen — sind die neugeschaffenen Grosskondensatorapparate, die mit grösserer Genauigkeit alles leisten, was wir eben wünschen und bisher nicht erreichen konnten. —

Schliesslich danken wir herzlich den Chefärzten, die uns freien Zutritt gaben, die Apparate ihrer Abteilungen zu untersuchen, den Technikern, die uns damit halfen, und besonders danken wir dem Leiter des *Biophysischen Instituts der Universität zu Kopenhagen, Herrn Prof., Dr. phil.* H. M. HANSEN für sein lebhaftes Interesse für die Untersuchungen und seine gütige Verfügestellung der Hilfsmittel seines Instituts.

ZUSAMMENFASSUNG

Die Arbeit beschäftigt sich einleitend mit strahlenphysikalischen Fragen, die besonders mit der Bildung der Kontraste bei der Aufnahme entstehen; es wird dabei u. a. hervorgehoben, dass es unter gewissen Umständen möglich sein wird, die bessere Strahlenausbeute der höheren Spannungen auszunutzen, weil die Streustrahlung im Diagnostikbereiche nur unwesentlich mit der Spannung steigt. Die Voraussetzungen hierfür sind teils ein geeigneter Film, teils ein besonders genau arbeitender Apparat. — Die Katho-

lenoszillographie als Messmethode wird beschrieben und ihre Brauchbarkeit für die Beurteilung der Leistungen der Diagnostikapparate wird durch die Wiedergabe einer Reihe von Oszillogrammen, die durch Messungen von üblichen Apparattypen gewonnen wurden, illustriert. Die Messergebnisse zeigen, dass Verbesserungen der Zeitregulierung und der Wiederholbarkeit der Leistungen bei vielen Apparaten zu verlangen sind. Die modernen Grosskondensatorapparate werden besprochen und ihre — jedenfalls theoretisch möglichen — Leistungsmöglichkeiten günstig beurteilt.

SUMMARY

The authors begin by discussing the radiophysical questions which specially arise in connexion with the contrast formation during exposure. They call attention specially to the fact that under certain circumstances it will be possible to utilise the larger ray output resulting from higher tensions, because the secondary radiation in the diagnosis area increases only slightly with the tension. The indispensable condition is, however, that a suitable film and an apparatus functioning with the highest degree of precision are employed. They next discuss oscillography as a method of mensuration, and illustrate the usefulness of that method as a mean of evaluating the performances of apparatus for diagnosis, by presenting a series of oscillograms taken by measurings of different current types of apparatus. The results show that many of these apparatus stand in need of improvements as regards time regulation and capacity for reproduction. In conclusion, they speak of the modern super-condenser apparatus, and express themselves favorably with regard to their — at least theoretically possible — efficiency.

RÉSUMÉ

L'ouvrage s'attache, en manière d'introduction, aux problèmes, ayant trait au rayonnement physical, que pose l'usage de matières contrastantes au moment de l'impression. On souligne, en cette occurrence, la possibilité, certaines conditions étant remplies, d'utiliser le rendement plus important qui accompagne une tension plus élevée; à la condition de se maintenir entre les limites usuelles, on n'assisterait qu'à une augmentation insensible du rayonnement secondaire avec celle de la tension. Un tel résultat se trouve assuré d'une part, par l'usage d'une plaque sensible spéciale et de l'autre, par l'emploi d'un appareil fonctionnant de manière satisfaisante. — L'oscillographie cathodique, employée comme méthode d'évaluation, donne ensuite lieu à une description. Une série d'oscillogrammes, dûs à des appareils de type courant, sert d'illustration aux services que l'on peut attendre de cette méthode dans l'évaluation de capacité des appareils diagnostiques. Les résultats quantitatifs indiquent qu'il serait nécessaire de soumettre un grand nombre d'appareils à une surveillance en ce qui concerne le réglage de temps et la reproductibilité. Les appareils à grand condensateur font enfin l'objet d'une mention. Les auteurs jugent favorablement et d'une manière toute théorique pour chaque cas les usages qu'il est permis d'en attendre.

FROM THE MEDICAL DEPARTMENT, AKADEMISKA SJUKHUSET, UPSALA (HEAD: PROF. C. BERGMARK), AND THE ROENTGEN DEPARTMENT, AKADEMISKA SJUKHUSET, UPSALA (HEAD: PROF. H. LAURELL)

THE ROENTGENOLOGICAL DIAGNOSIS OF SIDEROPENIC DYSPHAGIA[1]

(PLUMMER-VINSON's syndrome)

by

Jan Waldenström and *Sven Roland Kjellberg*

Among the different maladies of the oesophagus, very little attention has been paid to the socalled Plummer-Vinson syndrome or anemia with dysphagia even in the most extensive medical treatises. In the last edition of Handbuch der inneren Medizin by MOHR und STÄHELIN 91 pages are devoted to the maladies of the oesophagus but not a single word is said about this theoretically interesting and, from a practical point of view important malady. In the French literature also the condition seems to be practically unknown and although the voluminous work of TERRACOL »Les maladies de l'œsophage» is a very thorough treatment of the subject, nothing is even mentioned about this form of dysphagia.

It seems quite obvious that this outlook must be changed and chiefly for four reasons. 1) The malady is, beyond question, the most common of the diseases of the gullet and often gives dysphagia of the severest kind. 2) The results of our therapy are most favourable. 3) It seems probable that the cure of the condition called sideropenia (WALDENSTRÖM 1937), which is supposed to be the immediate cause of the dysphagia, may be an important prophylactic measure against cancer of the mouth and throat (AHLBOM et al.). 4) In the present paper the radiological features of the disease are described. This gives specific and objective signs as a basis for the diagnosis.

The real incidence of dysphagic symptoms among patients with essential hypochromic anemia (e. h. a.), also called iron deficiency anemia, is not easily determined. Quite a number of our patients have been sent to the

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radiological department because of symptoms in the throat, and it is possible that their disease would not have been diagnosed at this stage if it had not been for their dysphagia. The present material is, therefore, not suitable for a thorough and unprejudiced discussion of the incidence of dysphagia among patients with e. h. a. as it certainly gives an exaggerated representation of the cases with dysphagia. This problem will be investigated later by one of us in another connection. Among the present cases however, the large majority have come to the medical outpatients department because of their different typical symptoms of e. h. a. In the published material there are roentgenological changes in 54 among them 2 were men). Some patients (4) were sent to the hospital by friends or relations on the presumption that they might suffer from Plummer-Vinson's syndrome; in some others the condition was diagnosed from old case histories and the patients were brought to the hospital for roentgen examination. The rest of the cases (47) all came to the hospital spontaneously to seek aid for their complaints. They were seen during the years 1936, 1937, 1938 and 1939. If their number is divided by 3.5 this would give no less than 13 cases per annum. It may perhaps be of interest to compare these figures with those representing the frequency of other maladies of the oesophagus in this hospital during the same period. It is easily seen, that e. h. a. (P. V. dysphagia) is the most common of the maladies of the oesophagus.

Table 1.

Number of patients with typical roentgen changes.

	1936	1937	1938	1939 $^1/_1$—$^1/_8$	Total 3 years
Sideropenic dysphagia (P.-V.)	4	5	26	12	
Carcinoma oesophagi	5	7	5		17
» hypopharyngis			3		3
Cardiospasm	2		2		4

Another point is also rather interesting in this connection. The correct diagnosis of Plummer-Vinson's dysphagia has probably very often been missed especially during the first two years of the period. This is shown in Table I. The cases of cancer of the oesophagus and cardiospasm that have occurred in the same district during the period in question have certainly all been correctly diagnosed — and the figure for their frequency must be regarded as representative of the true incidence among the population.

The literature on Plummer-Vinson's dysphagia has been rather extensive since the symptom was first studied by KELLY and PATERSON, KELLY regarded the dysphagia as probably of nervous origin and was of the opinion that it was not a hysterical manifestation and had nothing

to do with the »globus». He found fissures at the corners of the mouth and observed muscular spasm and »taut membranous bands» in some cases. PATERSON observed the prominent association of dysphagia and superficial glossitis.

In 1922 Vinson published a paper called »Hysterical dysphagia» from the Mayo Clinic. Among his 69 cases there were 57 women. VINSON especially emphasized the common occurrence of severe anemia and pointed out, that PLUMMER observed the same syndrome already in 1914. Oesophagoscopy did not reveal any abnormality. MOERSCH and CONNER collected 65 cases with adequate data from the Mayo Clinic and confirmed VINSON's opinion that there was a great preponderance of women. They found achlorhydria in 8 out of 10 examined patients.

Subsequent authors (CAMERON, JONES and OWEN, GRAHAM and JOHNSON, HADEN, DAMESHEK a. o.) have collected cases and shown that the condition is not very uncommon, but they have not been able to add any information concerning the mechanism of the dysphagia. There has naturally been much discussion about this question. KELLY speaks about hyperesthesia of the sensory portion of the nervous arc as a result of irritation by foreign bodies, fissures etc. HURST has suggested a spasm caused by inflammatory changes in the nerve endings. He does not think that the anemia is due to a dietary deficiency. WITTS has observed that there might be a relapse of anemia even if the dysphagia remains healed. He therefore concludes that the dysphagia cannot be the cause of the anemia.

It seems as if the anatomical examination would give the best clue to the understanding of the malady but this has not been the case. It is very probable that the condition like so many other internal maladies is predominantly functional and does not give any very characteristic anatomical lesions. The material however is very scarce as the malady is practically never mortal. SUZMAN has described a case of a woman who died from a dilatation with perforation of the oesophagus and secondary mediastinitis. At the autopsy there was found hyperkeratinization of the epithelium with areas of atrophic degeneration. Another case with autopsy was recently described by MC GEE and GOODWIN. Their case was a woman 66 years old. Her story was somewhat atypical and the changes seem to have been of the most severe kind. After dilatation during four days she could swallow soft food and liquids. She was treated with iron but some days after she had left the hospital she died from a profuse gastric haemorrhage. At the postmortem examination there were found innumerable bleeding points not over 2 mm. in diameter in the mucosa of the stomach, duodenum and upper jejunum. There were recent haemorrhages into both adrenal glands. The findings in the oesophagus naturally were the object of chief interest. The inner surface of the

upper half of the oesophagus was hyperemic and had numerous irregular areas 4—10 mm. across, wich were denuded of mucosa. There was no demonstrable obstruction. Macroscopically mucous glands were plentyful in the inflamed upper end of the oesophagus. There were found numerous oval islands predominantly of lymphocytes with scattered plasma cells and polymorphonuclear leukocytes beneath the epithelium. The muscular wall was of increased width, varying from 3—6 mm. It was obvious that there was a definite chronic oesophagitis with erosion of the epithelial lining.

HOOVER gives the data of 17 cases. He mentions anatomical examinations of an oesophageal web removed with punch forceps. It consisted of 2 layers of mucous membrane with a small amount of fibrous tissue between them. HOOVER gives very thorough clinical details from his cases and was able to demonstrate webs in most of the cases that were oesophagoscoped. The treatment was mechanical dilatation and prescription of iron salts in adequate dosage. HOOVER especially points out that in a young woman without severe dysphagia the symptoms were relieved by antianemic treatment without dilatation.

From the foregoing it is obvious that the malady has chiefly been observed and diagnosed in the English-speaking countries. Very few cases seem to have been observed in France, and even German hematologists who have shown a special interest in this form of anemia as for instance SCHULTEN and HEILMEYER have only seen a very limited number of cases. As late as in 1938 THIELE und KÜHL published four cases of dysphagia with anemia and seemed to regard the condition as a great rarity. In Scandinavian countries this malady has received a great interest and the number of published cases is very large. (Cf. ZETTERQUIST, AHLBOM, WALDENSTRÖM, LUNDHOLM.)

We know very little about the roentgenological picture of this condition and most authors positively assert that nothing abnormal was found at the roentgen examination of the oesophagus. In some cases »a slight hesitation of the swallow was observed at the level of the larynx.» In 1938 WALDENSTRÖM described some cases with a typical and very characteristic roentgen appearance. In 1939 LUNDHOLM mentions that in 12 cases out of 14 nothing abnormal was found but in 2 cases, there was found a moderate constriction at the level of the first or second vertebra thoracalis. A detailed description is not given. We have therefore felt that it was necessary to investigate this question thoroughly from the clinical and roentgenological point of view.

Clinical picture

The syndrome that has been named after the authors PLUMMER and VINSON is most conveniently described as anemia with dysphagia.

The association of this dysphagia with other symptoms has been much discussed and it is obvious that most modern authors regard the dysphagia as simply a symptom of the socalled essential hypochromic anemia (cf. for instance LUNDHOLM).

The hematological characteristics of this form of anemia are the following: There is a pronounced decrease in the hemoglobin content of the blood. The number of erythrocytes is not correspondingly decreased, and *the anemia is* therefore *hypochromic*, i. e. with an index decidedly below one. Associated with this anemia there are found several *symptoms from the epithelial organs.*

The changes in the *nails* sometimes give the characteristical picture of koilonychia or spoon-nails. In such cases the nails are concave with upturned edges, very easily lamellating and brittle. It is common to find that the patient has suffered from these nail troubles for a very long time, sometimes even for decades. Very often the nails are not concave but show a definite tendency to split up and break at the free edge. They are usually soft.

Another symptom of great importance is a very *smooth* and varnished, usually reddish *tongue.* It is very often inflamed and then gives the patient severe discomfort. *Fissures* in the corners of the mouth (perlèche) are usually chronic even if they may heal for some time and then relapse.

WALDENSTRÖM has pointed out that *burnings and itchings in the vulva* are sometimes found in these cases and may disappear after intense iron-treatment. It does not seem improbable that also the mucous membranes of the genital organs may be the site of similar epithelial lesions as the mouth and throat.

Achlorhydria is often present, but cannot be regarded as an obligatory symptom. It is very often difficult to make a test meal when the dysphagia is severe.

One of the most characteristic subjective disorders in these cases is certainly the *difficulty in swallowing.* It is very common that the patients complain of pains and burning sensations when the morsel passes down behind the larynx or that it sticks at this level. If the morsel is small and soft nothing happens and it is unusual to hear about difficulties with the swallowing of fluids. The dysphagia is usually of long standing and sometimes the patients are not able to swallow any solid food and therefore get very thin and emaciated, a fact that might bring about a suspicion of cancer. It is therefore obvious that the clinical diagnosis of this form of dysphagia is not always quite simple even in typical cases.

WALDENSTRÖM (1938) has pointed out that he has seen a case with the typical signs of »dysphagia with anemia» but without anemia. He also observed several cases of koilonychia without anemia. Intense iron-

treatment gave a complete cure in all these cases. He put forwards therefore the hypothesis that the iron-deficiency does not affect the different organs of the body in the same way. The most common symptom is certainly the anemia. In other cases, however, there may be found e. g. achlorhydria with epithelial symptoms but without anemia. HALLÉN has collected some cases in which the achlorhydria improved under iron-treatment. We have been able to observe three cases of the so called P.-V. syndrome without anemia. In all of them the treatment with large doses of iron gave very quick relief.

Therefore we prefer not to speak of the P.-V. syndrome as this means dysphagia with anemia but shall use the new and correct term sideropenic dysphagia, usually combined with, but sometimes not associated with anemia and achlorhydria. This form of dysphagia ought to show a low serum iron value and is always improved or cured with an intense and prolonged iron-treatment. It is most important, however, that iron is given in really big doses and for a long time. The preparation we have used with great success is ferrous lactate in doses of one gram 3—4 times daily. Reduced iron if given with hydrochloric acid is also very effective. We have also seen a definite improvement after such preparations as Ferrofer and Ferrostabil in large doses, but it is quite certain that they are less effective than the first-named remedies. On the other hand, they are much better tolerated and are therefore of great value in some cases.

Our patients have usually improved very well on iron-treatment without any dilatation, but on the other hand there are several patients, who have shown a very prompt relief immediately after the passing of a bougie and this has started the subsequent complete cure with iron medication. It cannot be denied, that there are patients with long-standing sideropenic dysphagia who are not *completely* cured with iron treatment. We have not tried any systematic dilatation of these severe strictures as clinical experience has shown, that there is great risk for perforation of the oesophagus. We think it probable however that a very careful dilatation will be the best treatment of these cases in future if it is combined with large doses of iron.

Radiological picture

In order to get reliable pictures of the hypopharynx and the upper part of the oesophagus it is absolutely necessary to use a special technique. Only if the rules given below are strictly followed is it possible to get good pictures even in cases with very slight changes.

The patient is given a large morsel of a thick barium swallow. For this purpose Unibaryt has been found to be one of the best preparations. The opaque meal will have about the right thickness if six table spoonfuls of

Unibaryt are well mixed with two spoonfuls of water. When the meal is properly prepared it ought to flow down slowly like a thick sirup, when the spoon is taken out of the glass. It is desirable that the consistency is thick enough. Otherwise the hypopharynx and oesophagus will not be enough dilated when the patient swallows.

It is not easy to persuade these patients usually suffering from severe dysphagia to swallow the whole morsel at once or to swallow in absolutely the same way each time. It is therefore necessary to repeat the swallowings several times before making the picture and in this manner to teach the patient that he must swallow at once, when he is told to do so. The best plan is to control the process of deglutition before the fluorescent screen, until one is certain that the patient will swallow a morsel large enough to dilatate the oesophagus. This procedure has been completely successful except for the cases with very severe changes. The patients are then only capable of getting down a very small quantity each time, but the stricture is usually clearly seen on the film even in this way.

In order to get first class pictures it is necessary to make the exposure exactly at the moment when the swallow passes the lower part of the hypopharynx and the upper part of the oesophagus. In order to get these results it is easiest to count 1, 2, 3, 4 etc. in a loud voice and tell the patient to swallow just when you say 3. On the screen it is easy to see at what following number the morsel passes the right place. If this moment is found to correspond let us say to 6 in the counting, a picture is taken at this moment in a new trial and it is in this way quite simple to get good pictures. The method has long been used by LAURELL in order to photograph exactly the right phases during the act of deglutition.

Even for normal persons it is sometimes difficult to swallow exactly in the same way several times. It is therefore best to make a series of exposures with four pictures in frontal view and four in the sagittal. Usually some of the pictures will be taken at exactly the right moment. The best technique is to use ÅKERLUNDS diaphragm dividing a film measuring 18 × 24 cm in four parts. It is sometimes difficult for a patient with severe dysphagia to swallow as much as 8 big morsels. In such a case one has to be content with only four exposures, two in each direction. On the other hand it is quite important to get several pictures in order to make certain that a change in the outline of the gullet is really constant and not caused by a wave of contraction.

It is usually possible to observe the typical changes quite distinctly on the screen when following the passage of the swallow through the upper part of the oesophagus already at the first excercise with the patients. Only in those cases, which show very slight changes, is the diagnosis sometimes difficult on the screen.

Because of the very high speed at which the swallow passes down through the hypopharynx it is necessary to make as short exposures as possible in order to get really clear pictures. The required conditions are obtained when using 0.03 sec. as time of exposure. The pictures reproduced in this work have been taken with a rotating anode tube (Pantix) fed by a three-face generator Tridoros (Siemens) and all the exposures have been made with 300 mA and 72—75 kV for the lateral view and 80—85 kV for the frontal view. Since the last mentioned value, 80—85 kV, is the highest voltage which the tube used will safely stand, it was found necessary to increase the exposure time to 0.04—0.05 seconds in strongly built patients.

The normal picture of the hypopharynx and the upper part of the oesophagus usually shows a practically even surface on the lateral view. After a big swallow the lumen is usually 15—20 mm. wide (Fig. 1). In elderly patients there are sometimes to be seen rather flat, longstretched impressions on the posterior wall of the oesophagus, caused by marginal exostoses of the cervical vertebrae or prominent intervertebral discs. At the level of the cartilago cricoidea there is sometimes seen a certain applanation of the oesophageal lumen, caused by the lamina cartilaginis cricoideae. At the same level or just below it there is sometimes found a very narrow incision in the opaque meal most marked on the anterior wall of the hypopharynx or oesophagus. This very fine incision runs in a direction from above slightly obliquely downwards. It is probably caused by a very thin sometimes circular fold of the mucous membrane. With a correct technique this picture does not resemble what we find in Plummer-Vinson's dysphagia (see Fig. 3 and 4).

In 3 other cases there was found a very curious local protrusion of the posterior wall of the oesophagus at the place of Killians' oesophagus lip. All three patients were old women. When the swallow passes down through the opening of the oesophagus the tonic contraction of the musculus constrictor pharyngis normally relaxes and the oesophagus lip is not seen till after it has passed. In these 3 cases a protrusion resembling the oesophagus lip was seen during all phases of deglutition (see Fig. 5). The cause of this phenomenon has not been definitely found. There might possibly be a change in the reflex mechanism of swallowing in these old people resulting in a lasting tonic contraction of the sphincter. Two of the patients complained of some narrowness when swallowing. In one of them an oesophagoscopy was performed. The instrument was brought down to the entrance of the oesophagus without the least difficulty. The oesophagus lip was seen very clearly and was, all the time, in a state of tonic contraction in spite of the pressure exerted through the instrument. We owe these observations to Professor NYLÉN who kindly put them at our disposal. In normal cases there is a relaxation

of the musculus constrictor when the oesophagoscope is pressed against the entrance of the oesophagus or at deglutition or vomiting. In the three cases mentioned above nothing changed the appearance of the oesophagus lip and it was constantly seen to bulge into the lumen. Otherwise there were no pathological changes in the status of the patients.

On a frontal view the lumen is normally somewhat wider in the hypopharynx and continually narrows towards the oesophagus. The outlines are always smooth without any incisions. If the lower part of the hypopharynx is not sufficiently dilated there is seen a pattern of the mucous membrane behind the cartilago cricoidea. The folds of the mucosa are arcuate with the lateral convexity forming a picture that resembles a seal.

The roentgenological changes in the oesophagus occurring in sideropenic (Plummer-Vinson's) dysphagia are very typical. They are naturally to be seen more or less clearly according to the duration of the pathological process. The site of the lesion is usually found in the upper part of the oesophagus just below the cricoid cartilage (cf. Figs 6, 14 and 16) or quite exceptionally still lower down (Fig. 9), but never as far down as the crossing of the aorta. In most cases except the very advanced and severe ones the changes are most clearly or exclusively seen on the lateral view. There is in such cases found one, or very rarely several very thin (0.5—1.5 mm) defects in the opaque meal. Sometimes they are 5—6 mm. long, in other cases not more than a few mm., always if the gullet is well dilated pointing into the lumen at about right angles from the wall. The whole looks like a thin membrane (see Figs 6 and 8). Sometimes these changes are very slight. In such cases they are only seen on maximal dilatation of the oesophagus. Figs 13 and 14 demonstrate the paramount importance of this point. They are both from the same patient. In Fig. 13 the lumen has only been dilated to about half its maximal diameter and in this picture no pathological changes are seen. In Fig. 14 on the contrary there is seen a typical fold when the gullet is well dilated. Both pictures were taken on the same day. In somewhat more advanced cases these changes are also seen on a frontal view, with small incisions in the lateral outline (see e. g. Fig. 11). This arrangement agrees well with the pictures seen on oesophagoscopy with a thin semilunar membrane on the anterior wall stretching towards the lateral walls of the gullet. In advanced cases this incision is seen as a circular fold of the oesophageal walls (see Figs 12 and 15). Such patients usually have a severe dysphagia and the meal passes down very slowly and with great difficulty. There is sometimes seen a bulb-like swelling of the hypopharynx during a certain phase of the deglutition caused by a stagnation of the swallow before the stricture (see Fig. 15). A similar picture was published by HOOVER. In really inveterate cases there is also seen a broader cuff-like stricture below the membrane, certainly caused by an infiltration of the wall

(Fig. 10). Such a picture very much resembles what is seen in a carcinoma and the differential diagnosis may be very difficult.

The interesting problem of the connection between the Plummer-Vinson syndrome and carcinoma of the pharynx will be discussed later. With the exception of such advanced cases the wall of the oesophagus is usually smooth and the lumen of the gullet is the same below and above the stricture.

A question of special interest is the disappearance of these changes after proper treatment. There has been much discussion in medical literature about the best treatment for these complaints. From the beginning many authors regarded dilatation as the most effective therapeutic measure and it was even said that the anemia was the result of malnutrition caused by the dysphagia. The latter was regarded as of hysterical origin. The opinion now-a-days seems to be generally accepted that the administration of iron in sufficient doses relieves all the symptoms from the oesophagus. The passing of a bougie seems to quicken the recovery in some cases. It is always difficult to get an objective measure of the degree of dysphagia in a patient and it is usually only possible to get a general opinion that the patient feels much better. One of our patients however (a case quoted above as a typical sideropenic dysphagia but without anemia Fig. 17 and 18) observed that she was always obliged to continue her meal for a long time after the other patients in the ward had finished. After a 3 weeks' treatment with iron alone she was able to finish her meal as quickly as any other patient. As a rule, the patients feel a decided improvement in their condition already after a few days' treatment and the symptoms usually disappear completely in about a month's time. There are, however, inveterate cases in which proper treatment only gives partial relief. The roentgen picture in such cases is usually quite typical with a well-marked stricture and not only a fold of the mucous membrane (see Fig. 10).

It might be expected, that there would be a decided change in the roentgenological picture accompanying the subjective improvement. This does not always hold true. Even patients who have lost practically all their subjective discomforts still show absolutely the same picture as before. Even a very long time after the patient's anemia was healed and her severe dysphagia has disappeared the picture may remain the same. In Fig. 16 is shown one of our first cases (WALDENSTRÖM case V) with his typical roentgenological picture. She has been roentgen examined many times since that year and she has always shown the same picture. She has not had any definite dysphagic symptoms for several years. On the other hand there are several cases which show complete disappearance of the typical roentgen picture after treatment. It is very probable that the limited number (5) of patients, who have had typical symptoms of

dysphagia 1—3 years before the roentgen examination and who do not at present (after intense iron treatment) show any signs on the film are instances of healed sideropenic dysphagia.

In order to determine if the same roentgenological picture of the hypopharynx may be obtained also in normal cases or in diseased conditions other than sideropenic dysphagia, we have examined a control material of 145 cases and 64 patients with pernicious anemia. None of the controls showed any clinical epithelial signs of sideropenia and they were mostly treated in the medical wards under such diagnoses as sciatica, nephritis, neurosis etc. Some of the cases were normal persons suffering from no malady (nurses, medical students). This control material consists of women and men, all examined with the same technique as the others. In all these cases with the exception of four the roentgenological picture was perfectly normal. There was seen no trace of the change that is so characteristic even in the early stages of essential hypochromic anemia except one person (cf. later case III). The roentgen findings in the other three cases with an abnormal picture of the mucous membrane will be discussed together with the differential diagnosis. It did not resemble what we have found in P.-V. dysphagia and could not cause any serious diagnostic difficulties.

If the symptoms from the hypopharynx were only associated with the achlorhydria and not with the sideropenia they ought to be quite common in cases with pernicious anemia. We have, therefore, photographed all the cases of pernicious anemia under the age of 60 regularly attending the medical outpatients department in the Academic Hospital. This represents a very large part of the patients with p. a. in the whole province. In the large majority of cases there had been found a histamine refractive achlorhydria and the diagnosis had been confirmed through a sternal puncture and a typical reaction to liver extracts (campolon). The group comprises 64 cases. Among these were found two with definite changes of the Plummer-Vinson type but without subjective discomforts. All the others showed a perfectly normal picture. It therefore does not seem very probable that the achlorhydria alone may cause these changes. We also found other signs of sideropenia as a plausible explanation of the positive findings in the two patients with typical pernicious anemia.

The occurrence of dysphagia in patients suffering from pernicious anemia is very rare according to the literature on the subject. As far as I know there are only two cases published and they are now always quoted as instances of pernicious anemia showing Plummer-Vinson's syndrome. The case of CROSKERY was decidedly not suffering from p. a. The diagnosis was founded on a high value for the index (1.3), but this obviously depends upon a miscalculation as the index ought to be 0.8 instead! (hemoglobin 32, erythrocytes 2.2, index 0.8). Nothing is mentioned

about the effect of liver therapy. It seems obvious that this case was simply an essential hypochromic anemia. The other case is that of JONES and OWEN. Woman 50 years. *Anemic already as a girl.* For a long time dysphagia. Palpable spleen. Erythrocytes 1.4 mill. Hemoglobin 44, Index 1.5. With liver diet rapid improvement.

The first case is definitely not a pernicious anemia. In the second however the diagnosis seems to be very well founded but it is probable that the longstanding dysphagia was caused by a sideropenia preceding the pernicious anemia, as this latter malady is hardly ever seen before the age of 20.

The case histories in our own instances of p. a. with changes in the hypopharynx are briefly as follows (cases I and II):

Case I. Woman, 34 years. (See Fig. 20.) 2 children, 10 and 7 years old. For about a year dyspnoic; dyspeptic troubles; Status: Urobilinuria; Hgb. 55 %, erythrocytes 2.36. Index 1.2. Leucocytes 6,200. Differential count normal. Thrombocytes 266,000. No sternal puncture. Histamine refractive achlorhydria.

Treated with campolon 10 cc × 4. After a fortnight Hgb. 75 %, red cells 3.41 mill. After a month Hgb. 80 %, red cells 4.08 mill. The hemoglobin later increased to 90 %, erythrocytes to 4.8 mill. After an abortion in Febr. 1936 treated with iron. In May 1937 low values of hemoglobin (63 %), in spite of treatment with liver extract. Pregnant. End of July: in spite of iron treatment (ferrous lactate 1 gm. × 3) no real improvement. Normal blood values not until July 1938 after prolonged iron treatment (lowest index 0.8). On April 3:rd 1939 Hgb. 79 %, red cells 4.0 mill. No difficulty in swallowing. Never fissures in the mouth or koilonychia. Serum iron: about 50 γ %. On May 26th Hgb. 79 %, erythrocytes 3.5 mill. Serum iron 89 γ %.

Case II (See fig. 21): Woman 54. Admitted July 27th, 1935. Hgb. 40 %, erythrocytes 1.8 mill. Index 1.1. Thrombocytes 40,300. Temperature subfebrile. Histamine refractive achlorhydria. Smooth tongue. Urobilinuria. Sternal puncture: typical megaloblastic marrow. Icterus index 26 (MEULENGRACHT). After intense treatment with campolon (no iron) typical reticulocyte response. Dismissed after a month, Hgb. 60 %, erythrocytes 3.75 mill. After two months Hgb. 75 %, erythrocytes 4.47 mill. In the following years good results with the treatment. Febr. 23:rd 1939, Hgb. 87 %, erythrocytes 5.1 mill. April 14:th, Hbg. 83 %, erythrocytes 5.0 mill. The low value for the serum iron (40 γ %) seems to disclose a latent sideropenia.

Case III. Woman, 42 years. 10 living children, one abortion. Previous history of no interest. 13. 4. 39 roentgen examination of the oesophagus with typical changes as in sideropenic dysphagia. Never any subjective dysphagia. Nails always normal, never fissures in the corners of the mouth. Gastric acidity normal. Blood: no anemia (4.3 and 90 %). 22. 7. 1938 Blood: 4.6 and 95 %. The patient was re-examined on June 20th 1939. The roentgen picture of the oesophagus was the same as before. No other epithelial symptoms suggestive of sideropenia. Blood values normal. (80 and 4.5 mill.) Serum iron: 86 γ %.[1]

[1] The patient was re-examined on sept. 16, 1939. She now complains of definite dysphagia for some months. Also fissures in the corners of the mouth and tongueburn. Blood normal.

This case history is of considerable interest. Among the large number of control cases (209) showing no clinical signs, possibly indicating a sideropenia, there were only three showing radiological signs of dysphagia sideropenica. Two were the cases with pernicious anemia related above and the third is case III. It is naturally quite impossible to give any definite interpretation of the case but it must be noted, that changes in the roentgenological picture of the oesophagus might be found even in the absence of hematological or other epithelial symptoms. The value for the serum iron was also normal. We have seen several cases, however, where iron treatment has resulted in the disappearance of all objective and subjective symptoms of sideropenia except the changes in the oesophagus and it does not seem impossible that the patient might have suffered from a sideropenic condition in her earlier life. One of us (WALDENSTRÖM) has also seen patients with typical koilonychia without any signs of e. h. a. and with normal values for the serum iron. Such cases are probably very rare.

Differential diagnosis

It seems quite obvious from the foregoing that a large percentage of these cases have never been properly recognized as sideropenic dysphagia. We have seen one patient who came to the surgical out-patients' department complaining of a goitre and dysphagia. She was sent to the roentgen department to get a picture of the trachea and there the typical picture of a sideropenic dysphagia was found. The patient was not strikingly pale but her hemoglobin was only 57 %, she had typical fissures in the corners of the mouth, koilonychia and a histamine resistent achlorhydria! Quite a number of cases were sent to us with the suspicion of a malignant growth. The first case in which the typical radiological changes were discovered came to the surgical clinic with the diagnosis carcinoma of the oesophagus. She had not been able to eat any solid food for several years and was quite emaciated with a weight of only 46.7 kg. She was extremely pale with a hemoglobin of 30 % and erythrocytes 3.1 mill. From the roentgen picture alone it was not possible to exclude a malignant growth, but proper treatment with large doses of iron quickly improved her dysphagia and her general status. After 2 months' treatment she was able to eat quite well, her hematological and epithelial symptoms were all cured. Her weight had increased to 54.7 kg in six months. We have been in a position to follow this case with regular intervals. She still thinks that she has a narrow gullet and she is not able to swallow very big morsels. She has no real dysphagia however. Ever since the first treatment with iron her general status has been excellent, she has no anemia and there cannot be any suspicion of a malignant tumour. Her roentgenological status has remained unchanged!

Fig. 1.

Fig. 2.

Normal frontal and sagittal view of the upper oesophagus.

Fig. 3.

Fig. 4.

Fine folds of the mucous membrane in cases with no clinical signs of sideropenia. Cause unknown.

Picture 10 shows a case which may be difficult to differentiate from malignancy. It may seem tempting to use the other epithelial signs such as koilonychia and fissures as arguments in favour of the diagnosis sideropenic dysphagia. Of late, however, AHLBOM has investigated the problem of the connection between P.-V. dysphagia and carcinoma of the hypopharynx. He has found that a very high percentage of patients with

Fig. 5.

Fig. 6.

Fig. 5. Practically the same picture was seen in three old women without any clinical signs of sideropenia (see page 625. Cf. Schinz Lehrbuch der Röntgenologie Page 1611, 1939).

Fig. 6. Moderately severe sideropenic dysphagia. Woman 38 years old. For many years brittle nails. Status in Febr. 1938. Smooth tongue, fissures, koilonychia. Histamine refractive achlorhydria. Hemoglobin 41 %. E. 3.91 mill. After 6 months of intense iron therapy no symptoms. Slight secretion of HCl already after caffeine.

Fig. 7.

Fig. 8.

Woman 52 years old. For 6—7 years severe dysphagia of the severest type. Tongueburn. Nails concave. Fissures. Histamine refractive achlorhydria. Hypochromic anemia. After 4 months' intense treatment no dysphagia at all, no fissures, no koilonychia. Hemoglobin 110 %. Serum iron 57γ %.

A sister sideropenic dysphagia (roentgen). Healed with iron.

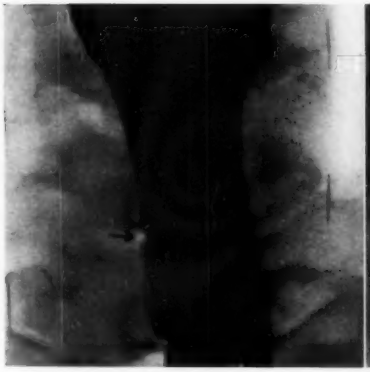

Fig. 9.

Fig. 10.

Fig. 9. Woman 47 years old. In 1934 anemia (55 %). Iron. In July 1938 relapse. Tongue smooth. Pains at deglutition. Nails brittle. Free HCl.: 25 fasting; 50 after caffeine. Hemoglobin 51 %, E. 4.8 mill. In the frontal view there are seen two lesions rather low down. Normal outline on sagittal view. Very unusual picture only found in this case.

Fig. 10. Woman 70 years old. Chronic dysphagia for several decades. Treated with radium and surgically for carcinoma of the bucca. Febr. 1939 Hemoglobin 82 %. E. 3.52 mill. Serum iron 38γ %.

Decided improvement with iron treatment. Probably a case of malignancy?

Fig. 11.

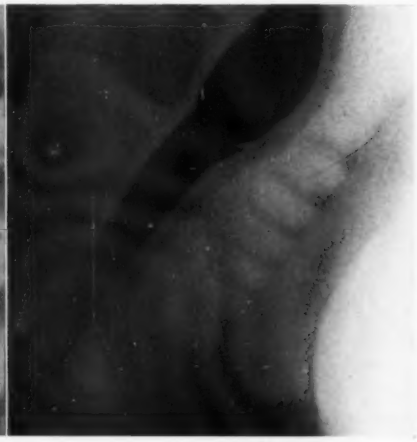

Fig. 12.

Woman 58 years old. For 15 years dysphagia. The last 10 years only fluid food. Very feeble. Totally invalid. Treated with small doses of iron since 1936. Status $^{12}/_{2}$ 38: Hemoglobin 82 %. E. 6.16 mill. Tongue smooth. No fissures. Koilonychia. Intense iron treatment gave no complete relief.

Fig. 13. Fig. 14.

Woman 56 years old. After resection of the stomach typical sideropenic anemia. The figures show the importance of a big swallow. When the oesophagus is only moderately distended the picture seems quite normal. A big morsel gives the typical picture. Only if the technique is absolutely correct it is possible to exclude the typical changes.

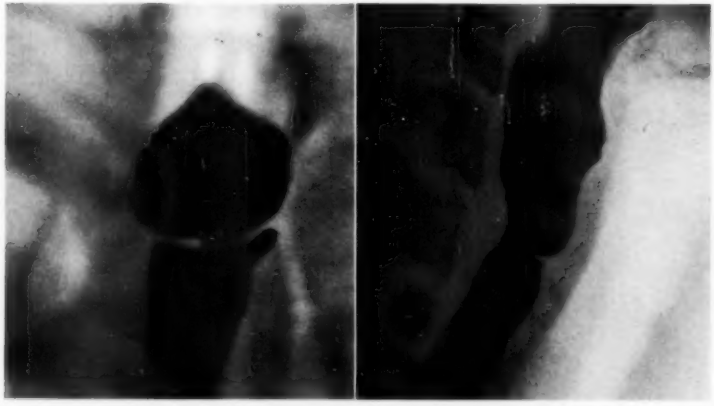

Fig. 15. Fig. 16.

Fig. 15. Man 34 years old. For many years dysphagia and brittle nails. Never treated for anemia. Hemoglobin 61 %. E. 4.7 mill. Severe koilonychia. Fissures. Serum iron 20γ %. All symptoms healed with iron treatment. This is a typical picture in a severe case.

Fig. 16. Woman 47 years old. In Nov. 1935 admitted to the Surgical Clinic with diagnosis: Tumor oesophagi. Status: Cachectic. Koilonychia. Fissures. Hemoglobin 25 %. E. 3.1 mill. Histamine-refractive achlorhydria. Treated in Med. Dep. with large doses of iron (6 gms daily). Jan. 1936 no dysphagia. Excellent condition. Hgb. 90 %. E. 5.14 mill. Re-examined 1936; 1937; 1939. Excellent status. No dysphagia. Roentgenological picture unchanged!

Fig. 17. Fig. 18. Fig. 19.

Figs 17 and 18. Woman 56 years old. For more than 30 years typical dysphagia. Nails brittle. Fissures. In 1929 roentgen of the oesophagus. »Narrowness at the level of the 6:th vertebra cervicalis». No diagnosis. In her youth »anemia». Never signs of anemia since then. Status $^1/_{10}$—38: Hemoglobin 93 %. E. 4.22 mill. Serum iron 34γ %. After a month with ferr. lact. 1 × 4 considerable improvement of dysphagia. Fissures healed. After four months practically no dysphagia. Serum iron 79γ %.

Fig. 19. Woman 56 years old. For a long time dysphagia. Fissures. Status: nails normal. Histamine resistent achlorhydria. Hemoglobin 77 %. E. 4.52 mill. Ferrous lactate 1 gm. × 5. After 3 weeks dysphagia much improved. Intense iron treatment for half a year. Subjective symptoms healed. After histamine free HCl. 25!

Fig. 20. Fig. 21. Fig. 22.

Figs 20 and 21. *Pernicious anemia* probably combined with sideropenia showing picture characteristic for sideropenic dysphagia (see page 629).

Fig. 22. Woman 37 years old. Since the age of 27 pernicious anemia; previously Grave's disease. Good results with Campolon! Also treated with iron. The index showed a tendency to remain low. Renewed iron treatment gave good results. Serum iron May 9th 1939: 27γ %. No dysphagia. The importance of the picture is not easy to judge. Similar changes were never seen in patients with typical e. h. a.

carcinoma of the hypopharynx suffer from the epithelial symptoms which are so common in e. h. a. Also a certain number of women with carcinoma in the mucous membranes of the mouth have a history resembling e. h. a. AHLBOM therefore regards e. h. a. as an important factor for the development of such carcinomas. This is naturally a fact that makes the clinical diagnosis still more difficult and we are quite convinced that there are patients in whom only the further development may show us the correct diagnosis. There are, however, certain features of the roentgen picture which may be of aid for the diagnosis as there are hardly any other diseases that may give a picture resembling sideropenic dysphagia. A very curious tonic contraction of the oesophagus lip has been discussed previously and it is hardly necessary to discuss the subject further especially as we do not know anything definite about its causes.

We have seen another accidental change in this region that does not seem to be in any way connected with sideropenic dysphagia. The picture is seen in Figs. 3 and 4. Two patients among the subjects taken as controls and not suffering from any signs of the P.-V. syndrome showed very much the same picture. It is clearly seen that it is caused by a fold of the mucous membrane. We have never been able to find the same changes in dysphagia sideropenica, and obviously it has nothing to do with this malady.

It is much more difficult to grasp the exact importance of a very curious picture seen in Fig. 22. We are not inclined to regard it as a form of sideropenic dysphagia, but the whole question is not easy to settle definitely until more cases of the same kind have been found.

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SUMMARY

The history and clinical picture of the socalled Plummer-Vinson syndrome (dysphagia with anemia) are summarized. One of the present authors has found several cases which showed the typical clinical picture even in the absence of anemia. The patients had previously received no iron treatment. The effect of iron medication was very favourable. The new term dysphagia sideropenica is therefore regarded as more appropriate. The incidence of this malady among the patients in the University hospital in Upsala is compared with the number of cases suffering from other common maladies of the oesophagus (Table I).

The technique necessary to obtain reliable roentgen pictures of the upper oesophagus is described in great detail. With this technique it has been possible to detect definite changes in every one of the 54 patients complaining of this type of dysphagia. 64 patients with pernicious anemia have been examined. Two of them showed the typical picture (their serum iron value was low and they were probably instances of true sideropenia). 145 control subjects were examined. Only one subject showed the changes and she was later found to suffer from dysphagia.

The changes sometimes disappear during treatment with iron but may also remain unchanged even after complete cure of the subjective symptoms. One such case has been followed since 1935 at regular intervals. The connection between dysphagia sideropenica and carcinoma of the hypopharynx is discussed.

ZUSAMMENFASSUNG

Die Geschichte und das klinische Bild des sogenannten Plummer-Vinsonschen Syndroms (Dysphagie mit Anämie) wird kurz beschrieben. Der eine von den Verfassern hat verschiedene Fälle beobachten können mit dem typischen Bilde dieses Syndroms aber ohne Anämie. Die Kranken waren früher mit Eisen nicht behandelt worden. Das Resultat der Eisentherapie war ausgezeichnet. Die Benennung Dysphagia sideropenica wird daher als die Zutreffendste neu eingeführt. Das Vorkommen dieser Krankheit in dem Krankenhausmaterial in Upsala wird mit der Frequenz von anderen Krankheiten des Oesophagus während der gleichen Zeit in Upsala verglichen (Tabelle I).

Die Technik um gute Röntgenbilder des oberen Teiles des Oesophagus zu erhalten wird sehr genau beschrieben. Nach den eben beschriebenen Vorschriften gelingt es leicht typische Veränderungen im oberen Oesophagus nachzuweisen und zwar bei allen 54 Kranken die an sideropener Dysphagie litten. 64 Krankee mit perniziöser Anämie wurden auch untersucht. Nur zwei von ihnen zeigten das charakteristische Röntgenbild. Ihr Serumeisenwert war niedrig und sie sind wahrscheinlich als Kombination von echter Sideropenie mit perniziöser Anämie zu betrachten. 145 Kontrollfälle wurden auch geröntgt. Eine Patientin hatte die Veränderung. Es wurde später gefunden dass sie auch an leichter Dysphagie litt.

Die Eisentherapie kann zuweilen die Oesophagusveränderungen zum Verschwinden bringen. Sie können aber sogar sehr lange nach erfolgreicher Therapie immer noch bestehen bleiben. Ein solcher Fall wurde seit 1935 in regelmässigen Abständen beobachtet. Der Zusammenhang zwischen der sideropenischen Dysphagie und dem Hypopharynxkarzinom wird besprochen.

RÉSUMÉ

L'histoire et l'image clinique du syndrome dit de Plummer-Vinson (dysphagie avec anémie) sont brièvement décrites. L'un des auteurs actuels a rencontré plusieurs cas montrant l'image clinique typique sans qu'il y ait cependant d'anémie. Les sujets n'avaient pas pris de fer auparavant. L'effet d'un traitement par le fer se montra très favorable. Le nouveau nom de dysphagie sidéropénique semble ainsi plus approprié. La fréquence de cette affection parmi les malades de l'Hôpital de l'Université d'Upsal est comparée à celle des autres maladies courantes de l'oesophage (tabl. I).

La technique nécessaire pour obtenir des images radiologiques fidèles de la partie supérieure de l'oesophage est décrite en détail. Par cette technique il a été possible de révéler des altérations chez chacun des 54 sujets qui se plaignaient de ce genre de dysphagie. 64 sujets souffrant d'anémie pernicieuse ont été examinés. Deux d'entre eux présentaient l'image typique (la teneur du sérum en fer était diminuée et ils étaient probablement en état de véritable sidéropénie). 145 sujets témoins ont été examinés. Un sujet seulement montra une altération et plus tard ce sujet fit de la dysphagie.

Les altérations disparaissent quelques fois durant le traitement par le fer, mais elles peuvent aussi rester tout à fait les mêmes après la disparition complète des symptômes subjectifs. Un tel cas a été suivi depuis 1935 à intervalles réguliers. La connexion entre la dysphagie sidéropénique et le cancer de l'hypopharynx est discutée.

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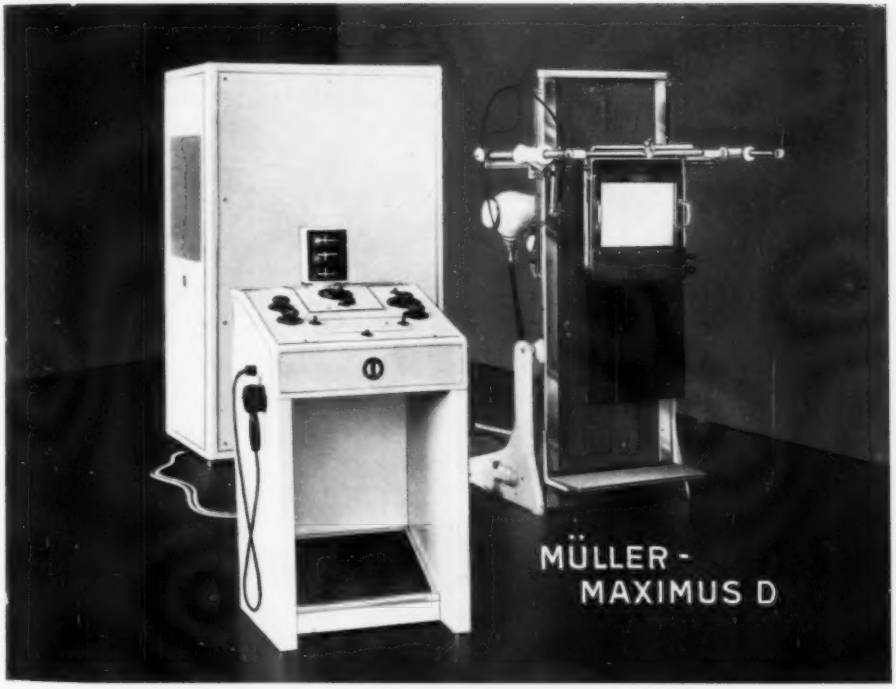

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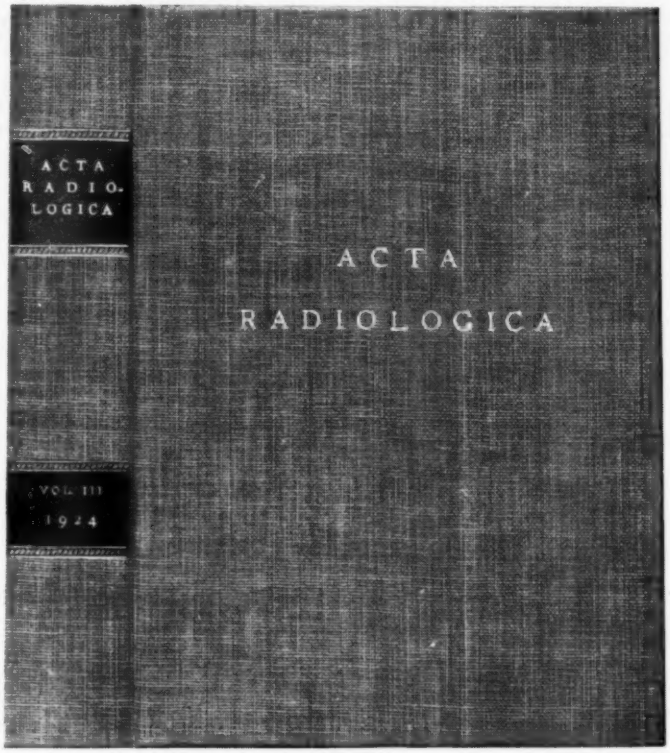

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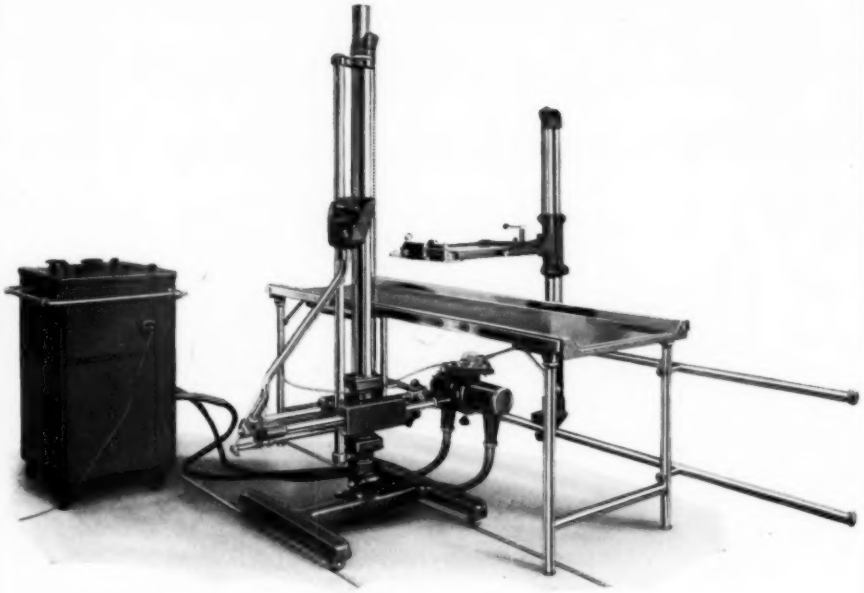

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